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OCULAR SYMPTOMS OF PSYCHOGENIC ORIGIN

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In everyday speech, and especially in slang, one encounters terms which indicate that common people recognize the influence of emotional response on bodily sensation. Such expressions as "eyes bulging" (with fear or desire), "kill with a look" or "I can't see him" are vivid examples of commonly known reactions associated with the eye.

A mechanism known in psychoanalytic theory as displacement consists in the moving of genital affects from a primary object or function to a secondary one. When displaced associations become attached with sufficient tenacity to the secondary object, the latter may assume the functions of the first and may eventually become a welcome substitute for the primary one in fulfilling certain emotional needs of the organism. Such substitutes are utilized in the unconsciousness of sleep or unconsciously in the waking state.

During waking hours substitutions springing from the unconscious as a result of a persistent and constant repression of unacceptable instinctual urges often come to light. These urges, particularly the sexual ones, are apt to continue at times when relief and satisfaction are normal, and when they are suppressed, but not relinquished, the denial of expression gives rise to a host of complaints referred to various bodily systems. These are in effect substitutions and displacements of the site of conflict to a guilty and/or weak organ.

Such substitutions may take the form of physical complaints ascribed to the gastrointestinal tract, producing nausea, vomiting and indigestion; to the eyes, producing "eyestrain," pain, blurring of vision and even blindness, or to the vascular system, producing palpitation, flushing and blushing. These symptoms are not necessarily confined to one system, but there may occur an interchange from one system to another or a combination of symptoms referable to two or more systems, such as tachycardia and diarrhea, or as indigestion and ocular symptoms in a person who "cannot stomach" and "cannot see" certain undesired situations. This is to be expected, inasmuch as the free floating sexual energy does not necessarily permanently invest the same nongenital system. The fact that these symptoms appear at times when the patient is partially satisfied by the expression of instinctual urges in masturbation and/or coitus is not a contradiction to the interpretation of these physical complaints as substitution reactions, for even at such times imperative moral and cultural remonstrances may prevent the patient from surrendering himself to sexual activity completely.

Experimentation in physiologic laboratories during the past thirty years (from von Bechterew and Pavlov to Cannon and the current experiments of Wolff and Masserman) has demonstrated the profound influence which emotion exerts on the action of the vegetative and sympathetic nervous systems. Each one of the great sensory apperceptive systems, among which is vision, can be directly affected by emotional stimuli to the extent that its function may be hampered or actually paralyzed. This general principle extends over a wide range of biologic reactions. Among these may be mentioned the temporary suppression of menstruation or urinary secretion, redistribution of the blood supply to the pelvic organs and changes in blood pressure and respiration. Physiologists recognize that a conditioning stimulus to any receiving organ may become so associated with the normal reflex as to bring about the same physiologic reaction.

In the psychologic study of neurotic symptomatology it has been found that organs which fail to perform their primary functions or are unconsciously condemned to inactivity have been utilized over an extended time in childhood for sexual gratification to an exaggerated and a disproportionate degree. In such cases all activities savoring of sexuality have been regarded with unusual disapproval by the parents. In many instances the person in disciplining himself unconsciously seems to adopt the tactics of the irritable mother who raps the child's hand when he takes something forbidden, or occasionally washes his mouth when he uses a "naughty" word, or puts him into a dark corner for peeping.

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"The eyes," quoted one woman, "are the windows of the soul." If the eyes mirror the soul, it is not surprising that the neurotic person, with his tendency to shift responsibility, should experience ocular difficulties—symptoms of tension—when he believes that his soul cannot stand inspection. Displacements of this type to the eye are facilitated through the great libidinous value which attaches to this organ, so habitually used for the silent expression and reception of emotion, especially the variations of love and hate, greed and scorn, pity and appeal.

In folklore and myth the eye is a symbol of the sun, the omnipotent eye of God, and the magic power of the evil eye is an old and still prevalent belief.[1] Symbolically, the single eye usually represents the female genital organ. For example, several years ago a prominent Philadelphia ophthalmologist underwent psychoanalysis because of a trembling of his hand whenever it was necessary for him to insert his scalpel into the pupil of the eye, although he was not troubled by tremor in any other phase of his operative work. This, however, constituted only the presenting symptom of his neurosis, the other, for him the more important, being impotence. Both symptoms were cured.

According to some interpretations, the single eye may also be symbolic of the phallus and the eyes together a symbol of the testicles, and the eyes and the nose collectively are symbolic of the male genitalia.[2] In the Lady Godiva and Oedipus legends blindness as a punishment for the guilty man has not only a direct but a symbolic implication. Especially in male patients, the fear of going blind or of having one's eyes "struck out" usually indicates a fear of (unconscious desire for) castration as a deserved punishment for violation of the prohibition of visual sexual curiosity.

A surprisingly large proportion of neurotic patients whose chief symptoms have taken the form of compulsions or obsessions other than those referable to the eyes, wear glasses or complain of trouble with their eyes. They describe their disability as "weak eyes"; blurred, limited or dazed vision; watering eyes, etc., and usually have sought the advice of the ophthalmologist. Occasionally some appreciation of the subjective psychic reaction to the visual difficulty creeps into the consciousness of the patient. One woman remarked, "I always see better when I feel mentally better." Another woman, when eyeglasses were prescribed for her in the vain hope that her headaches, due to a neurotic condition, might be alleviated, stated she felt that the glasses "formed a veil which permitted her to see without being seen," and a third patient under similar circumstances, adopted glasses because they seemed to form a shade which obscured the dark circles under his eyes, which he attributed to masturbation. Another patient, supposedly astigmatic, who had always been a coward, discovered as a boy that glasses protected him from fighting. When challenged, the wearer of glasses enjoyed momentary consideration from his opponent or from the by-standing group in the form of the comment, "Wait until he takes off his glasses." He then had an option of removing them or of hiding behind the excuse of not being able to see without his glasses. When this patient thought that he was about to be drafted into the war, on the assumption that his wearing glasses would gain immunity for him, he spent many hours reading the newspapers without glasses to convince himself how miserably he saw.

Trouble with the eyes is often consciously associated with too much use of the eyes, studiousness, scholarship and intellectuality, and this concept may extend itself to culture and refinement in general. Glasses then become the emblem of quiet respectability and a denial of sensuality and wickedness, especially excessive visual sexual interests. Who has ever seen a prostitute with spectacles or a chorus girl with a pince-nez on the stage? "Men make no passes at girls with glasses," remarked one wag. Then, too, one finds women who defer visiting the ophthalmologist because of the fear that the prescription of glasses may throw them into the respectable, asexual group. Often middle-aged men, regarding glasses as an evidence of approaching senescence (with its connotation of impotence), put off visiting the ophthalmologist as long as possible.

For all normal people looking is a strong element in sexual excitement, and the eyes are not ordinarily censured for lustful observation of persons of the opposite or of the same sex. However, when the eyes have been used for erotic gratification to an extent which the patient considers incriminating or incompatible with their proper function, subjective ocular difficulty may result. Many people suffer considerable difficulty either in wanting to see or in wishing to see more than is considered proper for them.

Similar visual trouble may even be carried into fantasy. A patient aged 34, who was still addicted to nightly autoerotism, began to wear glasses at the age of 18 because the optician told him that his eyes were a "little below par." He is an extremely shy person, one of whose greatest

1. For a survey of this aspect, see Greenacre, P.: The Eye Motif in Delusion and Fantasy, Am. J. Psychiat. **5**:553, 1925.

2. Jones, E.: Papers on Psychoanalysis, ed. 3, London, Baillière, Tindall & Cox, 1923, p. 363.

handicaps at present is his inability to enter (fear of entering) a store where there may be saleswomen because his eyes become fixed on their breasts. He remarked: "I am so self conscious that when I fancy sexual relations during masturbation, the picture of the woman is blurred. I am so ashamed that I cannot see a clear picture even in imagination." He himself appreciated that the blurring of the imaginary form prevented his passion from reaching its full height, thereby relieving him partially of the sense of guilt.

One must exclude from the category of persons to whom I call attention those who have only slight ocular complaints but for whom glasses have been prescribed on the vague hazard that in some mysterior manner they might relieve anxiety or an analogous nervous symptom. The facility with which neurotic wearers of eyeglasses are able to see clearly after their neuroses have been cured is presumptive evidence of the nervous origin of the ocular disorder. Mental adjustment renders the periodic adjustment of glasses superfluous.

Frequently patients report that the ophthalmologist at the time he prescribed glasses had himself expressed doubt as to the validity of the physical disability, and even as to the wisdom of his course in suggesting that the glasses be worn tentatively. Such a patient, after once adopting the glasses, seldom discontinues their use voluntarily because of the psychic comfort which he unconsciously derives from them. In fact, observed Derby,[3] "I firmly believe that opthalmologists produce more neuroses than they cure." Of course the opthalmologist does not produce the neurosis—it already exists. His treatments may tend to fixate it on this particular organ, as the orthopedist's braces may tend to fixate neurotic dissatisfaction on a weak back.

In this connection I shall report the case of a physician aged 40 whom I recently treated and who suffered from impotency, frequent bowel movements and a desire to cry. His ocular symptoms were as annoying as any of his other disabilities and began at the age of 22, at the same time as his impotence. At first the ocular difficulties appeared as photophobia, a burning sensation of the eyes, which usually came on when he was reading under or sitting in a room with artificial light. The burning sensation also presented itself when he was in the company of, or casually passed by, a woman with whom he had made unsuccessful attempts or when he was worried about a difficult case. It was associated with a sensation of tightening of the scalp.

The original trouble with his eyes, however, dated back to the age of 8 or 10 years, when he complained to his father that his sight was bad. The latter took him to an optometrist in the neighborhood, who prescribed glasses. He soon tired of them and discarded them, and he did not wear them again until the onset of the photophobia, during his first year in medical school, when glasses were again prescribed by one of the instructors at the college.

Between the ages of 21 and 25 he went from ophthalmologist to ophthalmologist, but the condition did not improve with any new set of glasses. Finally, he consulted Dr. George Derby, of Harvard Medical School, who convinced him that his complaint was not organic, but neurotic, by having him stare at an unshielded 100 watt bulb until his eyes began to burn. Dr. Derby then examined his eyes and, by having him look in a mirror, showed him that no redness was present. The patient then discarded glasses until about six years later, at the age of 31, when, because of frontal and occipital headaches, they were again prescribed, relieving his pain.. He did not use them for microscopic work.

He then wore glasses constantly until he entered the Army, at the age of 38, when he discontinued their use during the day. At the time of this report he wears them only when reading or at the movies. Except for mild myopia and astigmatism his eyes are in good condition. He has observed since he does not wear glasses regularly that if he is at a movie and forgets to put the glasses on, the mere thought that he had forgotten to wear them immediately brings about recurrence of the burning sensation in both eyes, which will continue for some time after he has left the theater.

Investigation of the possible psychic factor entering into his ocular symptoms indicated that the trouble began when, as a boy, he slept in the same room with his father and masturbated under the covers, always keeping a watchful lookout from the corner of his eye to see whether his father noted what he was doing.[4] The worry over masturbation caused him to become extremely self conscious in high school, with inability to concentrate and difficulty in making decisions. In college the impotency developed one year after the patient had contracted gonorrhea, and he had blamed it on his illness, even though he had been potent for a year after the attack. Essentially the impotency was associated with his conflict over masturbation. He then made every attempt, by the use of various mechanical devices, to desist from its practice and eventually managed to push the responsibility for masturbation into the unconscious, so that it occurred in his sleep rather than as a result of conscious fantasy. However, repression did not prove the solution of the conflict or the difficulty it produced in the eyes and elsewhere.

A word concerning the psychologic aspects of such treatments as the Bates's method seems timely. These procedures embody a great number of exercises, all of which are supposed to increase the power of sight. The directed ocular movements by suggestion give the patient the the right to see; in fact, they encourage the patient to use his eyes, so that self confidence is established in a person who, for reasons already mentioned, is insecure in use of his visual power.

The practitioner in any medical specialty can come to the conclusion that a symptom is a substitution reaction (conversion) only after a painstaking history and thorough physical examination. The differentiation between psychogenic symptoms and symptoms due to organic condi-

3. Derby, G. S.: Ocular Neuroses, J. A. M. A. **95**:913 (Sept. 27) 1930.

4. The reader is referred to analysis of the symptoms of eye rolling and masturbation by Briehl, W., and Kulka, E. W.: Lactation in a Virgin, Psychoanalyt. Quart. **4**:488, 1935.

tions often is the most vexing proposition encountered in medicine, and many a skilled diagnostician will confess that he has erred in both ways, e. g., in judging a condition as hysteria before the appearance of optic neuritis confirmed the diagnosis of cerebral tumor and vice versa.

Often it is advisable not to communicate the impression of the psychogenic nature of the illness at once to the patient. Indeed, to certain prejudiced, ignorant, fact-minded or old people it may be best never to state this opinion. If the patient is convinced that the physical examination has been minute and careful, the physician may impart the information that no organic basis for the complaint is demonstrable. Once the physician has decided to tell the patient that his symptoms are neurotic, it is probably best for him to desist from physical manipulations, such as applications to the eyes, throat or nose. If in doubt he should at least discontinue topical applications, and the like, until he is convinced that the patient is not amenable to direct psychotherapy. When suggestive therapy must be employed, a minimal use of physical procedures is permissible as a vehicle for the suggestion.

At present the baffling mystery of the relation of emotional stimuli, the reaction of the vegetative system, glandular secretion and congenital or acquired organic weakness is not solved. The exact mechanism by which so-called emotional impulses are transmitted by the sympathetic nervous system still remains unknown. Science can do no better than permit the physiologist, the endocrinologist, the allergist and the psychologist each to pursue investigations without restriction in his special field, in the hope that some day the manner of physiologic operation may be revealed. The clinical psychotherapeutist must confine himself to the presentation of such facts and interpretations as seem valid in accordance with his method. The soundness of the bases on which he works rests largely on recognition of their validity by the patient and on disappearance of the symptoms when the inhibitions which necessitated conversion phenomena no longer operate.

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DISCUSSION

DR. MARK J. SCHOENBERG, New York: I shall limit my comments to my experience in the field of glaucoma and shall begin in an unorthodox fashion by stating my conclusions first.

1. States of anxiety and emotional upsets may bring about a rise in intraocular pressure in a certain percentage of cases of glaucoma.

2. A glaucomatous crisis may be prevented by avoidance of emotional upset.

3. The diagnosis of glaucoma is incomplete and the treatment less effective without a survey of the patient's emotional life.

4. A positive history of emotional upset in a patient with glaucoma does not always imply a causal relation between the two.

Is there any evidence that there really exists a psychosomatic basis for glaucoma? Can emotional upset cause a rise of intraocular pressure? Some of the evidence is circumstantial; some is based on facts. Experiments on animals and clinical experience have shown that emotional upsets are accompanied by profound bodily and mental changes, distributed throughout the organism. The more or less sudden graying of the hair and the functional disturbances of the musculature and of the nervous, gastrointestinal, cardiovascular, respiratory and endocrine systems are evidences of the range of disturbances accompanying emotional upsets. Careful consideration of all these bodily changes leads one to believe that the sympathetic, parasympathetic and endocrine systems are the means, or meditators, by which the emotional energy, originating in the upper and lower emotional levels of the brain, is driven to and discharged in every part of the organism.

The sensory organs, especially the visual system, also participate in the emotional display. With respect to ophthalmologic evidence, one could mention the following observations:

1. Bulging of the eyes, widening of the interpalpebral fissures, lacrimation, enlargement of the pupils, spasm of retinal vessels, weakening or spasms of accommodation and temporary strabismus.

2. The long-standing clinical experience that acute glaucoma may develop during or after emotional disturbances like grief, panic or worry.

3. The decrease of intraocular pressure in some patients on whom the tonometric measurement is made twice in succession at about a five minute interval. This is due to the patient's mental relaxation following his awareness that the application of the tonometer is not "an operation" or a "painful procedure."

4. The increase of intraocular pressure in some patients who when undergoing scotometric examination for the first time become apprehensive under the strain of mental concentration, especially at the tangent screen.

5. The lack of response to miotics of some patients during periods of emotional stress, followed by amelioration of the response after elimination or appeasement of the mental upset.

I feel that as methods of measuring the intraocular pressure become more accurate and refined, one will be better able to ascertain, and even to measure, the relation between psychic upsets and the disturbances in the mechanism of intraocular pressure in patients with glaucoma.

GONIOSCOPIC CORRELATES OF RESPONSIVENESS TO MIOTICS

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That the qualitative, as well as the quantitative, response to the local administration of miotics varies greatly with the various types and stages of glaucoma is a common, if not daily, experience in the practice of ophthalmology. It has been the subject of extensive discussion in the literature, although during the last decade more stress seems to have been laid on the effect of new miotics, and not so much on their specific effect in certain types of glaucoma. Since the advent of gonioscopy, progress seems to have been made with regard to the sharper definition of some forms of glaucoma, and it appears worth while to review a series of cases of conservatively treated glaucomas with the aim of determining to what extent gonioscopically defined types of glaucoma possess the quality of responsiveness to miotics, or, more specifically, whether responsiveness to miotics has a gonioscopic correlate. It would obviously be of practical value if such a correlate could be shown to exist.

This study was made on a series of cases encountered in the glaucoma clinic of the Illinois Eye and Ear Infirmary.

SO-CALLED PRIMARY GLAUCOMAS

Classification of the so-called primary glaucomas on the basis of the gonioscopic observations is not altogether new. Long before the advent of gonioscopy one can find in the literature references [1] to the occurrence among the so-called primary glaucomas of two different mechanisms concerned with the part played by the angle of the anterior chamber. From clinical and pathologic observations the deduction had been made that in some cases closure of the entrance to the angle of the chamber by contact between the iris and the anterior wall of the angle constitutes the primary pressure-elevating mechanism (mechanism 1), whereas in other cases the intraocular pressure becomes, and usually remains, elevated for a long time before any portion of the angle becomes closed (mechanism 2).

Through the introduction of gonioscopy as a routine method of examination in cases of the glaucomas, the occurrence of these two mechanisms has been confirmed on innumerable living eyes. Gonioscopically the difference in the mechanisms is so striking that it has been suggested as a criterion for classification of the so-called primary glaucomas. The mechanism of the first type, which has been greatly elucidated by gonioscopic observations, is well expressed by the term "iris block," or "narrow angle" glaucoma (Barkan). Little is known about the mechanism of the second type, to designate which the names "wide angle," "trabecular" and "senile" glaucoma have been suggested. The awkward term "non-narrow angle glaucoma" expresses most precisely the present state of knowledge, or rather ignorance, concerning this form of glaucoma. Since in many cases of this type of glaucoma the probable site of the disturbance was somewhere in the trabecula–canal of Schlemm apparatus, I have used the term "T-S-group." At present, however, it seems best to use the noncommittal term "wide angle glaucoma." Glaucoma of the iris block or narrow angle type may now be defined as a form of glaucoma in which the normal intraocular pressure balance becomes disturbed only when the entrance to the angle of the anterior chamber, which is unusually narrow in cases of this type, becomes temporarily or permanently closed by angle-crowding factors or, later, by peripheral anterior synechias. Of the angle-crowding factors, the role of mydriasis —physiologic or emotional, or that induced by drugs—has been established. The presence of forward movement of the root of the iris during accommodation, which has been described and photographed by Sugar [2] in normal persons and in patients with narrow angle glaucoma (both

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All tonometric readings referred to in this paper were obtained with tonometers of the Schiøtz type, with use of the scale of 1924.

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1. Peters, A.: Das Glaukom, in Graefe, A., and Saemisch, T.: Handbuch der gesamten Augenheilkunde, ed. 3, Berlin, Julius Springer, 1930, pp. 243-244.

2. Sugar, H. S.: Am. J. Ophth. **24**:851, 1941.

under 45), is at variance with the classic view (Hess [3]), but this movement is probably an important angle-crowding factor in eyes with a narrow entrance to the angle. Other mechanisms by which acute increases in the volume of the ciliary body may occur have been studied by Sugar.[2] There is no definite evidence to indicate that the unusual narrowness of the entrance to the angle or the occurrence of angle-crowding factors is not a physiologic phenomenon. The physiologic growth of the lens may bring about narrowness of the entrance to the angle in eyes of a certain build.[4] This narrowness of the entrance to the angle, which is usually, but not always, associated with over-all shallowness of the anterior chamber, is the chief requisite for and is preexistent to elevation of intraocular pressure. In making this statement, I by no means intend to deny the occurrence of true glaucomatous shallowing of the anterior chamber, in the sense that the latter has been brought on by a glaucomatous process. On the contrary, phases of elevated intraocular pressure in cases of wide angle glaucoma may be associated with a measurable decrease in depth of the entire anterior chamber.

On the basis of the foregoing definition, the diagnosis of narrow angle glaucoma is justifiable if, in an eye with narrow entrance to the angle, a parallelism can be observed between partial or total closure of the entrance and elevations of intraocular pressure. The recognition of this parallelism may become difficult, or even impossible, if the angle is too narrow for gonioscopic study. If, in such a case, the intraocular pressure is normal for long periods without the use of miotics, it is probable that the function of the trabecula–canal of Schlemm apparatus is undisturbed and that temporary elevations of intraocular pressure are caused by partial or complete blocking of the entrance to the angle, although this blocking cannot actually be seen gonioscopically. However, if the entrance to the angle is so narrow that gonioscopic examination is impossible and a miotic is necessary to keep the intraocular pressure within normal limits, one cannot say with certainty that in the particular case the glaucoma is of the narrow angle type because there the miotic may act by enhancing the function of the trabecula–canal of Schlemm mechanism or in some other pressure-reducing, but not angle-freeing, way.

Without the aid of the gonioscope it is difficult to make with certainty the diagnosis of narrow angle glaucoma. Shallowness of the chamber alone does not constitute proof that the glaucoma in the case in question is of the narrow angle type because there is no absolute parallelism between the over-all depth of the chamber and the width of the entrance to the angle. In a case in which the chamber is very shallow the gonioscope may reveal a narrow, but open, entrance through which most, if not all, of the trabecular area is visible. If at this moment the intraocular pressure is elevated, the narrowness of the angle cannot be the cause of the disturbance of the pressure balance, and the glaucoma, although the angle is narrow, cannot be of the narrow angle type. On the other hand, gonioscopic examination may reveal narrowness, and even partial closure, of the entrance to the angle in a case in which clinical examination did not suggest it.[5] Strictly, only glaucoma of the type described by Serr,[6] in which the intraocular pressure rose sharply only when the pupil dilated beyond a certain threshold value, could be classified as glaucoma of the narrow angle type without gonioscopic confirmation of the relation between temporary closure of the angle and increases in intraocular pressure.

The same considerations apply to the diagnosis of wide angle glaucoma. If the intraocular pressure is elevated in an eye in which the angle is wide and roomy and in which there is not the slightest possibility of an iris block, the diagnosis is easy. The wide angle mechanism may, however, also be present in eyes with slight or pronounced narrowness of the entrance to the angle. In cases of the latter condition the glaucoma may be a combination of the wide angle and the iris block type.

The principal difference between the two types of so-called primary glaucoma may be concealed by the development of peripheral anterior synechias, that is, an anatomic union between the iris and the anterior wall of the angle, an event which is secondary in both types of glaucoma. In the narrow angle type peripheral anterior synechias are more likely to form at an early stage, whereas in the wide angle type they may form late or never develop.

With the aforementioned limitations, and perhaps others not yet fully realized, the concept of

3. Hess, C. V.: Accommodation in Man, in Bethe, A.; von Bergmann, G.; Embden, G., and Ellinger, A.: Handbuch der normalen und pathologischen Physiologie, Berlin, Julius Springer, 1929, vol. 1, p. 145.

4. Kronfeld, P. C., and McGarry, H. I.: Am. J. Ophth. **27**:1023, 1944. Sugar, H. S.: ibid. **25**:1341, 1942.

5. To what extent examination of the eye with the slit lamp without a contact lens furnishes clues to the width of the entrance of the angle is being studied by Dr. Paul Sternberg, of this institution.

6. Serr, H.: Ber. u. d. Versamml. d. deutsch. ophth. Gesellsch. **45**:22, 1925.

a distinction between a narrow angle and a wide angle mechanism operative in the glaucomas appears to be sufficiently well founded, and of enough practical value, to warrant its employment at present as a principle of classification. In the use of such a classification, it should be kept in mind that only the narrow angle mechanism is fairly well understood and that little is actually known concerning the mechanism of the probably heterogeneous group of glaucomas of the wide angle type. Besides, it should be remembered that cases of glaucoma of the narrow angle type make up only a small percentage of all cases of the so-called primary glaucomas.

GLAUCOMA OF IRIS BLOCK TYPE

A large number of cases of true narrow angle glaucoma occurred in my series of cases of conservatively treated, so-called primary glaucomas. In some of these cases the patient had been followed for as long as ten years. The average period of observation was approximately two and a half years. Several principles of classification of these cases were suggested: the degree of the "angle predicament"; the presence or absence of peripheral anterior synechias; the stage of the glaucoma, whether early or advanced, as judged by the amount of loss of visual fields or atrophy of the optic nerve, and the presence or absence of congestion or incompensation. In presentation of my observations it seemed convenient to divide the cases into three groups: (1) cases in which there were no peripheral anterior synechias; (2) cases in which a severe acute attack existed, and (3) cases in which peripheral anterior synechias, but not a severe acute attack, were present.

Group 1.—These cases had in common the presence of a severe angle predicament and the occurrence of spontaneous or artificially induced phases of elevated intraocular pressure. In the large majority of cases these phases were associated with corneal edema, congestion of varying degree and subjective symptoms of blurring of vision, presence of halos around lights and ocular discomfort of varying severity. In a small percentage of cases, however, the phases of elevated intraocular pressure caused no edema of the cornea or any subjective symptoms. Whether or not corneal edema, with its attending subjective symptoms, occurred in a given case depended partly on the height of the intraocular pressure reached during these phases; to some degree it appeared to be a characteristic feature of the individual eye, not related to the severity of the preexisting angle handicap.

In the majority of the cases in group 1 no permanent damage had been done to the optic nerve, the optic disk and the visual fields showing no glaucomatous changes. Only in about 25 per cent of cases had the sum total of the glaucomatous episodes been great enough to produce changes in the disk and the visual field without leading to formation of peripheral anterior synechias. The retina or the optic disk may have been unusually vulnerable in these cases.

The incidence of spontaneous elevations of the intraocular pressure before treatment was begun varied greatly in the cases of group 1. In some cases a short stay in a semidarkened room, such as a movie theater, was sufficient to bring on a rise in intraocular pressure. In other cases only unusual circumstances, such as severe emotion, shock or a combination of several of the angle-crowding factors, caused an elevation of pressure. Finally, there were cases in which only artificial, moderate or maximal dilatation of the pupil alone or combined with other factors caused a disturbance of outflow of the intraocular fluid. A parallelism between the angle predicament as estimated gonioscopically or goniometrically and the "ease" with which a rise of intraocular pressure occurred appeared to be present in some, but not in all, cases of group 1. In other words, of eyes with the same degree of narrowness of the angle, some had experienced almost daily upsets of the intraocular balance, whereas in other eyes unusual combinations of circumstances were necessary to bring on an elevation of intraocular pressure. The excursions of the pupil, or rather its variability under the conditions of normal life, may have been the deciding factor.

In general, the cases of group 1 proved to be responsive to miotics. Since the increases in intraocular pressure associated with narrow angle glaucoma occur irregularly, and not in accordance with any definite diurnal pattern, the doses of the miotic must be evenly distributed over the waking hours of the patient, with special consideration for stays in a dark or in a semidarkened room, for periods of prolonged close work and for anticipated severe emotions. Because of the miosis present during sleep, it is unlikely that any rises in intraocular pressure occur or begin while the patient is asleep. I have looked for the negative (mydriatic) phase of the action of pilocarpine which has been reported[7] but have failed to observe it twelve, sixteen or twenty-four hours after a single instillation of a 2 per cent solution of pilocarpine

7. Dixon, W. E., and Ransom, F., in Heffter, A.: Handbuch der experimentellen Pharmakologie, Berlin, Julius Springer, 1924, vol. 2, pt. 2, p. 765.

nitrate. Such a negative phase might be dangerous in cases of narrow angle glaucoma.

The amount of the miotic necessary to prevent increases in intraocular pressure varied somewhat, but most patients remained free of subjective symptoms and retained normal fields and a normal appearance of the disks with 3 or 4 daily instillations of a 2 per cent solution of pilocarpine nitrate. For many persons this dose may have constituted overtreatment, as was shown with some of the younger patients, in whom the instillation of a 2 per cent solution of pilocarpine nitrate caused blurring of vision which lasted from one-half to one and one-half hours. In several such patients two instillations of a 0.5 to 0.2 per cent solution of pilocarpine nitrate twice during the working day controlled the situation.

A number of patients are getting along well by using a miotic only on the development of slight subjective symptoms. This mode of treatment may be reasonably safe for intelligent patients, in whom even slight increases in intraocular pressure have been proved to be regularly associated with subjective symptoms. Since there is always the possibility that the ocular condition will get out of hand, my associates and I at the glaucoma clinic do not recommend this mode of treatment.

It is probable that the miotics exert their beneficial effect on the glaucoma of the narrow angle type largely through their miotic action.[8] In the cases in which only one or two instillations of a weak solution of pilocarpine are sufficient, the action of the drug may be said to be that of an antimydriatic, rather than that of a miotic. The possibility that the miotics have other important actions in cases of this type of glaucoma cannot be denied. Observations, made originally by Serr and confirmed by Sugar [2] and me, on a critical threshold value for the size of the pupil above which the intraocular pressure starts to rise make it probable that the size of the pupil is the most important factor in the mechanism of acute obstruction of the angle associated with narrow angle glaucoma; the miotics, therefore, probably act chiefly as miotics.

In cases of the highest degrees of angle predicament, pronounced or maximal miosis may be necessary to keep the entrance to the angle open. In these cases more frequent application of the stronger miotics, such as physostigmine, neostigmine or carbaminoyl choline chloride, is called for. In the majority of cases of this kind, although the angle was too narrow to be explored to its vertex, the factor of iris block in production of the glaucoma was made at least highly probable by the fact that during the phases of low intraocular pressure a larger portion of the zone posterior to the line of Schwalbe was visible than during the phases of high intraocular pressure. In the few cases in which not even this parallelism could be observed, the combined mechanisms of iris block and wide angle may have been present.

In most cases of group 1 the angle predicament did not seem to grow worse during the period of observation, and no tolerance to the miotics was acquired. In a few cases in which more frequent instillations of stronger miotics were required as time went on, I had the impression that the angle was becoming narrower. Thus, it seems to be characteristic of glaucoma of narrow angle type that no true tolerance to miotics is acquired.

Group 2.—During the acute congestive phases of glaucoma of the narrow angle type the relation between the gonioscopic picture and the responsiveness to miotics is difficult to establish. Until recently the presence of corneal edema has been considered an insuperable obstacle to gonioscopic study. While methods have been devised to clear the cornea, such as massage (Larsson [9]) and instillation of glycerin (Cogan [10]), it is not to be expected that gonioscopy will give unequivocal information with regard to the extent of the existing synechias. During the acute phase the angle is closed either by contact or by the formation of true synechias. It is unlikely that even after the cornea is completely cleared the distinction between contact and the presence of synechias could be made with certainty. It is even doubtful whether there exists a sharp line between mere contact, on the one hand, and the presence of synechias, on the other. Furthermore, it is reasonable to assume that among the synechias there are reparable, or "breakable," ones, on the one hand, and irreparable ones, on the other. Thus, even if the gonioscopic examination were done under ideal conditions of visibility it is unlikely that it could give, during the phase of acute congestion, a clear idea of the extent of the irreparable synechias.

A different situation prevails after the acute attack has subsided under the influence of miotics. Favorable response to miotics in such cases has, according to Sugar [11] and Ban-

8. Hruby, K.: Arch. f. Ophth. **141**:517, 1940.

9. Larsson, S.: Acta ophth. **17**:297, 1939.

10. Cogan, D. C.: Am. J. Ophth. **26**:551, 1943.

11. Sugar, H. S.: Gonioscopy and Glaucoma, Arch. Ophth. **25**:674 (April) 1941.

gerter and Goldmann [12] and in my experience, invariably been associated with the gonioscopic picture of at least a partly open angle. The smallest sector of open angle that was compatible with normal intraocular pressure was 70 to 90 degrees of arc. In the majority of cases a larger sector of the angle was patent. Thus the relation between the gonioscopic picture and the responsiveness to miotics in cases of acute congestive glaucoma of the narrow angle type could be expressed as follows: A miotic is successful in relieving an acute attack if over an area of the angle of at least 90 degrees the contact or the synechias are "breakable." Since their breakability could not have been recognized during the acute phase, it is doubtful whether gonioscopy during this phase could give any information of prognostic value.

Failure of the miotic, however, does not mean that the peripheral anterior synechias are of such nature and extent that an angle-freeing operation on the order of an iridectomy will not be sufficient to restore the normal pressure balance within the eye. As has been shown in several of our cases, under the condition of acute incompensation iridectomy is more effective as an angle-freeing measure than use of even the strongest miotics. Surgical removal of the root of the iris over an area of from 20 to 30 degrees of arc, plus the opening of other portions of the angle to the extent of at least 45 degrees—which probably occurs after the iris block is partly relieved by the iridectomy—has resulted in the permanent patency of an adequate portion of the angle in cases in which instillation of miotics alone or combined with osmotherapy had failed. The failure of miotics, therefore, does not give any clue as to whether a simple angle-freeing operation or formation of a fistula is necessary in a given case of acute congestive glaucoma of narrow angle type. The surgeon will be guided largely by the duration of the present attack and by other clinical data from which the total effect of previous glaucomatous episodes with consequent formation of peripheral anterior synechias can be estimated. Since there is always the possibility that the iridectomy will be insufficient, many surgeons routinely add to it an incarceration of the iris.

Group 3.—Under this heading fall the cases of glaucoma of the narrow angle type in which, as a result of one, several or many attacks, self limited or terminated by administration of miotics, peripheral anterior synechias of varying meridional and circumferential extent have developed. In principle, the same rule applied to these cases as to the cases of group 2. If three fourths or less of the circumference of the angle was closed, there still existed the possibility of normal intraocular pressure without the use of miotics. But in most cases in this group the patient was dependent on miotics in the sense that even under the conditions of normal life phases of elevated intraocular pressure occurred frequently and regularly. The response to miotics was good, and no tolerance was acquired over periods of several years. Administration of a miotic at regular intervals during the day was the treatment recommended by the glaucoma clinic in these cases of partly closed angle.

Entirely, or almost entirely, closed angles were encountered in cases of unsuccessful irridectomy for acute congestive glaucoma. In these cases miotics were administered in large or maximal doses before a second operation was resorted to. In no instance did the miotic bring the intraocular pressure down to the normal level, but careful observations, including those made during control periods, showed that the miotics possess a slight pressure-lowering effect, even in cases of a completely, or almost completely, closed angle. The actual reduction of intraocular pressure was not greater than 10 to 15 mm. and was, therefore, of little practical value. Tolerance to the miotic in these cases was definitely acquired after a few weeks. Osmotherapy was more effective than the use of miotics in any form or concentration. Similar observations were made on eyes which had not been operated on and in which repeated mild or severe attacks had led to complete closure of the angle. Again, miotics had a definite, but slight and insufficient, effect.

At this point it might be well to remind the reader that, strictly speaking, the diagnosis of complete closure is impossible to make by gonioscopic examination alone. There is always the possibility of filtration through small openings in the synechias. Intraocular fluid from the anterior chamber may enter the iris anterior to the anterior border of the synechias, filter through the iris and, by-passing the synechias, gain access to open pockets in the angle. Observations made on pathologic specimens suggest the possibility and feasibility of such a mechanism.[14] Because of this possibility, no conclusion can yet be drawn with regard to the point of action of the miotic in cases of a completely "closed angle." There are still at least two main possibilities,

12. Bangerter, A., and Goldmann, H.: Ophthalmologica **102**:21, 1941.

13. Footnote deleted by the authors.

14. Elschnig, A., in Henke, F., and Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1928, vol. 2, pt. 1, p. 880.

namely, increased transfer of fluid out of the eye by means of the trabecula–canal of Schlemm apparatus and reduction of the rate of fluid transfer —blood or intraocular fluid—into the eye.

Finally, brief reference should be made to the cases of narrow angle glaucoma in which after one or several attacks the pupil has become "frozen" in maximal or submaximal dilatation. Partial or complete peripheral anterior synechias, atrophy of the iris and complete fixation of the pupil are characteristic features. To what extent the fixation is anatomic, that is, due to necrosis and subsequent fibrosis of the iris, and to what extent it is neurologic, that is, due to paralysis of the local ganglionic apparatus, I have not been able to decide in any of my cases. None of the miotics (except histamine, which has not been tried) produced any measurable change in the size of the pupil. In these cases if the posterior anterior synechias occupy less than three fourths of the angle circumference, the fundamental rule for narrow angle glaucoma still applies. The intraocular pressure remains normal without the use of miotics as long as sufficient portions of the trabecular area are gonioscopically visible. When these portions become closed off, as the result of forward movement of the root of the iris, the intraocular pressure becomes elevated. These elevations are prevented and controlled by the use of miotics. The mechanism of this action is not clear. There is no possibility that the miotics can act as such, since the pupil is completely fixed. Their cyclotonic action would, according to Sugar,[2] constitute a pressure-elevating, rather than a pressure-lowering, factor. It would be logical to conclude that the effect of the miotics on the glaucomatous eye is complex and that a pressure-lowering action is the result of the preponderance of the pressure-lowering factors.

GLAUCOMA OF WIDE ANGLE TYPE

The apparently primary glaucomas in which the presence of the iris block or the narrow angle mechanism has been excluded are classified here under the heading of wide angle glaucomas. The following gonioscopic observations have been described in these cases: (1) pigment deposits of varying degree within the corneoscleral trabecula; (2) signs of abnormal opacity (veiling, or *Verfilzung* [Bangerter and Goldmann [12]]), of the uveal portion of the trabecula; (3) abnormal filling phenomena of the canal of Schlemm after paracentesis of the anterior chamber or massage of the eyeball (Kronfeld, McGarry and Smith [15]), and (4) peripheral anterior synechias in the late stages.

Friedenwald [16] suggested that we attempt to correlate the responsiveness to miotics with the abnormal filling phenomena just indicated. This work is now in progress, but no definite conclusion has been reached. The abnormal opacity of the uveal portion of the trabecula, which was described by the two Swiss authors,[12] has been of rare and indefinite occurrence in my series of cases, so that no clear relation between its presence and the responsiveness to miotics could be established. A survey of my series of cases, however, revealed clearly the absence of any consistent relation between the intensity of pigment infiltration of the trabecula and the responsiveness to miotics. Good and lasting responses to the miotics were observed with about the same frequency in eyes with heavily pigmented trabecular zones as in eyes with lightly pigmented ones. In the cases of far advanced glaucoma of wide angle type, with or without peripheral anterior synechias, there was less responsiveness to miotics than in the cases of early glaucoma without synechias. Except for this feature, the gonioscopic picture of glaucoma of the wide angle type permitted no prognosis with regard to the efficacy of miotics.

GLAUCOMAS SECONDARY TO IRIDOCYCLITIS

Under this heading are comprised cases of chronic or recurrent anterior uveitis in which ocular hypertension has occurred in the absence of pronounced activity of the inflammatory process. Not included are cases of seclusion of the pupil with partial or complete iris bombé and cases of acute elevation of intraocular pressure occurring simultaneously with mild recurrent cyclitis.[17] I wish to repeat here a statement in my discussion on the paper "Uveitis with Secondary Glaucoma," by Fralick and associates.[18]

> One of the many annoying and exasperating features of iridocyclitic secondary glaucoma is the extreme variability and irregularity of the disease. If in a case of [inactive or slightly active] iridocyclitis the intraocular pressure becomes elevated, it is difficult to predict whether this is going to be just a brief episode or whether this will prove a permanent secondary glaucoma, resistant to all forms of medical and surgical therapy which are at our disposal. Even the gonioscopic findings do not always enable us to tell with certainty whether the anatomy of the chamber angle

15. Kronfeld, P. C.; McGarry, H. I., and Smith, H. E.: Am. J. Ophth. **25**:1163, 1942.

16. Friedenwald, J.: Personal communication to the author.

17. In my opinion, this condition represents a clinical entity.

18. Fralick, F. B.; Cooper, J. H., and Armstrong, R. C.: Tr. Am. Acad. Ophth. **47**:92, 1942.

has been altered to such an extent that the function of the normal channels of outflow is permanently or irreparably impaired.

The gonioscopic findings in cases of glaucoma secondary to iridocyclitis vary within wide limits. The principal features are inflammatory debris and pigment on and within the trabecula and in the angle, nodes or specific granulomas and their sequelae, peripheral anterior synechias of more or less characteristic appearance, postinflammatory membranes and neovascularization. In correlating the gonioscopic picture with the responsiveness to miotics, the erratic and irregular course of glaucoma secondary to iridocyclitis had to be reckoned with. Repeated control periods were necessary to determine the general course of the intraocular pressure without medication. In some cases the tension curves, after having been followed for several days, proved so irregular that it was impossible to determine whether a drop in intraocular pressure after the administration of miotics could be considered a response to the latter or a vagary of the glaucoma.

In 7 cases a fairly regular tension curve was obtained without medication, and the response to miotics could be established. Correlation of this response with the gonioscopic picture revealed no specific relation. In general it seemed to be true that the miotics were less effective in the cases of more severe and extensive alteration in the angle. Beyond that it was not possible to predict the efficacy of the miotics from the gonioscopic picture.

GLAUCOMA CAUSED BY PERIPHERAL ANTERIOR SYNECHIAS RESULTING FROM DELAYED RESTORATION OF THE ANTERIOR CHAMBER AFTER CATARACT EXTRACTION

This form of glaucoma was studied gonioscopically by Sugar [19] and by me in collaboration with Grossman.[20] The extent of the peripheral anterior synechias is largely dependent on the length of time the root of the iris had been in contact with the anterior wall of the angle. The gonioscope reveals three variations of the anterior edge (toward the anterior chamber) of the adhesions, namely: a solid, sharply demarcated line; a fuzzy, scalloped zone, and multiple thready, filamentous adhesions.

The condition of the anterior border layer of the iris seemed to determine the type of edge present. The dense border layer without crypts, such as is found in most dark brown eyes, tended to give rise to adhesions with solid line edges. On the other hand, the delicate blue or grey iris usually forms synechiae characterized by fuzzy edges.[20]

The latter type of synechias, especially the multiple filamentous adhesions, give rise to less severe disturbances of the intraocular pressure balance than do the synechias with solid line edges.

Most cases of glaucoma of this group respond well to miotics. If the patient is first seen during a phase of very high intraocular pressure, dramatic drops, from 70 to 20 mm., may be observed to occur within two or three hours after the repeated administration of a 0.25 to 0.5 per cent aqueous solution of physostigmine salicylate. If the peripheral anterior synechias are extensive and with solid line edges, the tonometric readings usually vary from 40 to 60 mm. To keep the intraocular pressure at a normal level, strong miotics have to be administered at short intervals. In my experience physostigmine has been specifically effective in these cases. With the same number of applications, 0.25 to 0.5 per cent aqueous solutions of physostigmine salicylate have a stronger pressure-lowering effect than has pilocarpine nitrate in 4 to 6 per cent concentration used as a solution or ointment. Often, however, the beneficial effect of the miotic is not exerted longer than two months, after which administration of stronger solutions at shorter intervals must be resorted to. Thus, a tolerance seems to be acquired within a relatively short time, and in most cases of this type surgical treatment is finally required.

In cases in which less extensive and more filamentous peripheral anterior synechias are present the disturbance of the intraocular pressure balance is less severe, the tonometric readings varying from 25 to 40 mm. In these cases the pressure responds well to small or moderate doses of pilocarpine nitrate (2 per cent aqueous solution twice to three times a day), and no tolerance is acquired. While the eye is under the influence of miotics and in a state of normal intraocular pressure, the gonioscopic picture is not appreciably different from that obtained during phases of high intraocular pressure. Thus, the miotics reduce the intraocular pressure without altering the appearance of the synechias which are responsible for the elevation of intraocular pressure. To what extent these observations permit conclusions with regard to the point of action of the miotics in these cases will be the subject of a separate publication. Here, it may suffice to state that peripheral anterior synechias of limited extent, especially those of filamentous nature, produce relatively mild disturbances of

19. Sugar, H. S.: Am. J. Ophth. **23**:853, 1940.

20. Kronfeld, P. C., and Grossman, E. E.: Tr. Am. Acad. Ophth. (1940) **45**:184, 1941.

the intraocular pressure balance, disturbances which are as a rule permanently and easily controlled with miotics. The gonioscopic correlate of lasting responsiveness to miotics in cases of this type of glaucoma is a type of peripheral anterior synechia that is not too extensive and not too solid. The term "correlate" is used here in the broader sense of regular co-occurrence of the two phenomena, and not in the sense of a direct relation of cause and effect between the synechias and the responsiveness to miotics.

GLAUCOMA ASSOCIATED WITH CAPSULAR EXFOLIATION

In eyes with senile capsular exfoliation, irrespective of whether or not glaucoma is present, the gonioscope reveals particles of exfoliated capsular material deposited on both walls and in the vertex of the angle, in addition to pronounced pigment infiltration of the trabecula. A review of the cases in which this gonioscopic picture was associated with chronic glaucoma not of the iris block type showed clearly that the quality of responsiveness to miotics was present to a very low degree. The situation is well expressed in the conclusion reached by Gradle and Sugar [21] that "many of these [patients] do not respond well, or only temporarily, to miotics." In the files of the glaucoma clinic of the Illinois Eye and Ear Infirmary there is no record of a case in which the glaucoma could be controlled with miotics for as long as four years, as Irvine [22] had observed. This survey failed to yield unequivocal data with regard to the efficacy of the miotics as a prophylactic, or glaucoma-preventing, measure (Mueller [23]; Linn [24]).

SUMMARY

Gonioscopic correlates of responsiveness to miotics are shown to exist with the various phases of glaucoma of the iris block type, with the glaucoma caused by peripheral anterior synechias due to delayed restoration of the anterior chamber after cataract extraction and with the glaucoma associated with capsular exfoliation. In cases of so-called primary glaucomas not of the iris block type and of glaucoma secondary to iridocyclitis there is no apparent relation between the gonioscopic picture and the efficacy of miotics.

21. Gradle, H. S., and Sugar, H. S.: Am. J. Ophth. **23**:982, 1940.

22. Irvine, R.: Exfoliation of Lens Capsule (Glaucoma Capsularis), Arch. Ophth. **23**:138 (Jan.) 1940.

23. Mueller, H. K.: Klin. Monatsbl. f. Augenh. **98**: 653, 1937.

24. Linn, J. G.: Exfoliation of the Anterior Capsule of the Lens, Arch. Ophth. **21**:717 (April) 1939.

ABSTRACT OF DISCUSSION

Dr. H. Isabelle McGarry, Chicago: In cases of glaucoma of the narrow angle type, Dr. Kronfeld mentions the frequent presence of pronounced angle predicament without peripheral anterior synechias. In many cases in which the angles in the two eyes appear exactly the same, one eye may show a notable increase in tension, requiring miotics or surgical treatment or both, and the other eye may not show hypertension unless provoked by mydriasis. In the clinic my associates and I consider the eye with the normal pressure, normal fields and normal disks as preglaucomatous and prevent mydriasis with miotics.

I, also, have been unable to come to any definite conclusion regarding glaucoma of the wide angle type and glaucoma secondary to iridocyclitis.

We have also encountered the occasional case of glaucoma secondary to extraction of the lens in which the angle appears almost blocked by fairly dense synechias and the pressure rises shortly after operation, to be controlled only by a strong miotic, such as physostigmine, and later becomes normal and remains so without the use of miotics, even though the synechias appear unchanged.

Dr. Kronfeld avoids the term "glaucoma capsulare" and speaks of glaucoma associated with exfoliation of the capsule of the lens. Gonioscopic study bears out this point, since in many cases the amount of trabecular pigmentation and exfoliation my be almost the same in the two eyes, the disk, pressure, visual fields and vision being normal in one eye while the other eye shows far advanced glaucoma, as demonstrated by a study of the fields, tension and fundus. This type of glaucoma as a rule is uncontrolled by miotics.

Dr. John M. McLean, New York: Gonioscopy, while of restricted usefulness to the pure clinician, is a valuable tool in the hands of the clinical investigator. It has already demonstrated the fallacies of the theories which explain chronic simple glaucoma on the basis of anterior peripheral synechias. It has also shown that synechias are the result, not the cause, of chronic congestive glaucoma. It has demonstrated that synechias are the cause of previously unexplained glaucoma following cataract extraction. It has explained some of the failures and unexpected results of operations.

Dr. Kronfeld has reported the results of his attempt to correlate the gonioscopic observations with the effect of miotics on glaucomatous eyes. He has demonstrated a correlation between miosis itself and reduction of tension in certain cases of glaucoma of the "iris block" type. This does not hold true in all cases of this type, nor does it hold for other forms of glaucoma. More-

over, all who practice routine gonioscopy in the study of glaucomatous eyes find many cases in which there are open angles and elevated intraocular pressure which responds to use of the miotic drugs.

Similarly, in some patients with glaucoma of the open angle type introduction of mydriasis as a provocative test fails to produce a rise in tension. I can cite a family with three generations of congenital absence of the iris. Several members of this family had glaucoma. Although there was no iris sphincter and no possibility of producing miosis, miotic drugs caused a definite fall in intraocular pressure.

The whole question of the method of action of parasympathetic drugs in cases of glaucoma must be reconsidered. When it was believed that all forms of glaucoma were produced by embarrassment of the filtration angle of the iris, miosis alone seemed to explain the tension-reducing effect. It is now known that the old theories were wrong. It is understood why iridectomy is ineffectual in most cases of glaucoma. However, it is still noted that drugs which produce miosis reduce tension in many eyes in which there is no iris in the angle to be drawn out. Do such drugs act by contracting the longitudinal fibers of the ciliary muscle and exerting tension on the scleral spur? Do they thus increase the efficiency of Schlemm's canal? Do they act through ciliary spasm to reduce the blood flow to the ciliary processes and reduce the formation of aqueous? Do they have a direct vascular reaction and thus affect the fluid balance? Answers to these questions are not yet available, but without gonioscopy the questions might not even have arisen.

Dr. C. W. Ascher, Cincinnati: If gonioscopic examination does not supply correlates of responsiveness to miotics, an efficiency test of the exhaust system can be performed in cases in which, under favorable conditions, the outflowing aqueous humor may become biomicroscopically visible. This occurs in the *Randloops* or in a vein originating from the corneal limbus or, still farther away, in a vein leaving a scleral emissarium. These vessels, which contain aqueous humor, either clear or mixed with blood, have been called aqueous veins.

Compression of the recipient vessel of an aqueous vein produces either expulsion of blood from the blocked section of the vessel or expulsion of clear fluid from the particular aqueous vein. In glaucomatous eyes the latter effect is the rule, indicating weak elimination of aqueous humor from this aqueous vein, and possibly from the whole exhaust system, probably due to a transient or, in other cases, to a permanent narrowing of the immediate outlets of the canal.

Miotics, as well as surgical intervention can reestablish a more vigorous flow in favorable cases, and then expulsion of blood from the blocked section of the vessel may be seen during the compression test performed on the aqueous vein. These results parallel in a certain sense those of Dr. Kronfeld. Biomicroscopy and gonioscopy complement each other to give a better understanding of the exhaust system of the eye.

Dr. Peter C. Kronfeld, Chicago: I agree with Dr. McGarry that there are eyes which show the angle predicament to a high degree and still glaucoma is provoked only by unusual circumstances. One may see the same angle predicament in the eyes of the same patient. Increased intraocular pressure may be provoked in one eye by a short stay in the dark, whereas the other eye, with the same narrowness of the angle, remains undisturbed. I still think that the excursions made by the pupil during the course of normal life may have something to do with the ease with which attacks occur in such cases.

"Glaucoma associated with capsular exfoliation" should, for the present, be the preferred term. It is true that ophthalomologists are eagerly looking for clues to the mechanism of wide angle glaucoma, but I am not sure that the presence of exfoliated material in the angle always means impaired function of the trabecula.

Dr. McLean's comments have greatly clarified the situation. When I said that with certain types of glaucoma the miotics act as miotics, I was referring only to glaucoma of the narrow angle type. Some of the observations which I presented here indicate that a mechanism not related to the angle or to the iris underlies the action of miotics in cases of wide angle glaucoma. Just what that mechanism is I do not know, but my associates and I are following a few leads at the moment which look rather promising.

I did not quite understand Dr. Ascher's comment. I suppose that the aqueous veins he has described show certain alterations under the influence of miotics. About such alterations I know little.

Illinois Eye and Ear Infirmary.

KERATITIS NEUROPARALYTICA

CORNEAL LESIONS FOLLOWING OPERATIONS FOR TRIGEMINAL NEURALGIA

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Degenerative changes in the cornea following lesions of the trigeminal nerve have long been observed and are well known. Magendie demonstrated them experimentally in 1924 and postulated the presence of specific trophic nerve fibers in the trigeminal nerve, the function of which was to regulate the metabolism of the tissues. Since Magendie's time many other etiologic theories have been presented, the important factors which have been emphasized being vasomotor disturbance, trauma and desiccation.

The concept of trophic fibers has now been generally rejected, but some control of cellular activity by the sensory nerves is recognized. Thus, Duke-Elder expressed the belief that lack of normal peripheral antidromic activity causes disturbance in metabolism and is the chief etiologic factor in neuroparalytic keratitis. It is also admitted that vasomotor disturbance, trauma and desiccation are of importance as secondary factors.

The outstanding characteristic of all cases of this disease is anesthesia of the cornea, which is due to paralysis of the trigeminal nerve. This paralysis may result from a lesion in the gasserian ganglion or in the peripheral nerves or in the supraganglionic tracts. Lesions may be caused by pressure from tumor or by tuberculoma, tabes, syringomyelia, basal meningitis or trauma. Any of the surgical procedures evolved for the treatment of trigeminal neuralgia will produce serious trauma to the nerve. This paper is primarily concerned with a study of the corneal lesions following such operations. Two major types of corneal lesions may follow surgical intervention for trigeminal neuralgia, namely, neuroparalytic keratitis and exposure keratitis.

NEUROPARALYTIC KERATITIS

Neuroparalytic keratitis is a disease entity well recognized and described in textbooks of ophthalmology so that it is unnecessary to discuss it in detail here. Its characteristic features may be summarized as follows:

- Vasodilatation
  - Conjunctival congestion
  - Congestion of iris
- Changes in corneal epithelium
  - Central edema
    - Fine punctate stippling
    - Vesiculation
  - Erosion of vesicles
    - Multiple erosion
    - Coalescence
  - Exfoliation of epithelium
  - Ulceration
- Sequelae
  - Hypopyon
  - Perforation
  - Staphyloma
  - Panophthalmitis
  - Leukoma

The forms of neuroparalytic keratitis may be classified as follows: (1) parenchymatous, with absence of ulcerations; (2) superficial, a benign form, with areas of desquamation; (3) ulcerative, with a tendency to regression and moderate corneal damage, and (4) persistent. The lesions of the last form are refractory and advance to the periphery, with little tendency to recover.

An important characteristic is the variation in the severity of the process. Minute vesicles develop, which may go on to ulceration, or, to quote Duke-Elder,[1] "occasionally the process does not progress materially further, and resolves itself into a picture resembling superficial punctate keratitis or recurrent erosions." For the purposes of study, I shall divide corneal lesions of the neuroparalytic type as (1) severe, with gross loss of epithelium, and (2) mild, with only diffuse vesicles, which erode and stain with fluorescein. The mild form I should like to describe as diffuse epithelial vesiculation. The lesions of the severe form were treated more radically; there was more severe corneal damage, and visual loss was pronounced. The lesions of the mild form, or diffuse epithelial vesiculation, were treated conservatively; corneal damage was slight, and vision was not seriously affected. Corneal anesthesia was present in both forms, and, in my opinion, there is no difference in the

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Read before the Section on Ophthalmology at the Ninety-Fourth Annual Session of the American Medical Association, Chicago, June 14, 1944.

1. Duke-Elder, W. S.: Text-Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1938.

pathogenesis. In many cases of the severe form the disease was seen in the mild stage, and it is impossible to state in any individual case that a lesion seen at this stage will not progress to the more severe form.

EXPOSURE KERATITIS

Exposure keratitis, the second of the two major types of corneal lesions following operations on the trigeminal nerve, is a degenerative condition due to desiccation resulting from lagophthalmos. The characteristics of this condition may be summarized as follows:

1. Lesion of lower part of cornea
   - Desiccation
     - Horizontal band; punctate haziness
   - Fissures
   - Exfoliation
   - Haziness of stroma
   - Keratinization of epithelium
2. Sequelae
   - Secondary infection
   - Ulceration
   - Hypopyon
   - Perforation
   - Panophthalmitis

The lesions are progressive if not treated. Treatment consists of protection of the cornea.

In the cases seen in this study the condition was the sequela of facial paralysis, a complication of intracranial operations. The resulting paralysis of the orbicularis muscle furnishes the etiologic factor in the development of this disease. Anesthesia is not a necessary factor; it may or may not occur. However, if it exists, the causative factors of neuroparalytic keratitis are present concurrently, and the probability that the latter condition will develop is increased.

RELATION OF CORNEAL LESIONS TO OPERATIONS FOR TRIGEMINAL NEURALGIA PERFORMED IN UNIVERSITY HOSPITAL FROM 1927 TO 1943

Types of Operations.—Operation for trigeminal neuralgia was performed in 878 cases by members of the department of neurosurgery of the University Hospital during the period from 1927 to 1943. Corneal lesions followed in 18 per cent of the cases. These lesions varied from the minor changes of diffuse epithelial vesiculation to severe neuroparalytic ulcers, with loss of the eye.

The following operations were performed:

Injections of alcohol into the peripheral branches (supraorbital, infraorbital, maxillary and mandibular) of the trigeminal nerve, 16 cases.

Injections of alcohol into the gasserian ganglion, 4 cases.

Sections of the sensory root of the gasserian ganglion (rhizotomy), 858 cases.

Many patients had one or more injections of alcohol into the peripheral branches before undergoing a rhizotomy. Corneal lesions were not observed to follow such injections. Injections of alcohol into the gasserian ganglion were few. Most of the operations, therefore, were either complete or incomplete rhizotomies, as follows:

Subtotal rhizotomy involving the maxillary and/or the mandibular branches only (466 operations). This procedure should not interfere with the function of the first branch, and corneal sensation should be preserved.

Incomplete rhizotomies involving the ophthalmic branch (32 operations). Corneal anesthesia necessarily will result.

Complete rhizotomies (360 operations). The entire sensory root is severed. Corneal anesthesia will follow.

A total of 392 ophthalmic roots were severed, and a total of 878 operations were performed.

TABLE 1.—*Incidence of Corneal Lesions Following Operations for Trigeminal Neuralgia (University Hospital, 1927-1943)*

Corneal Lesions	Number	Diffuse Epithelial Vesiculation	Neuroparalytic Keratitis	Exposure Keratitis	Number	Incidence, Percentage
Injection of alcohol into branch of fifth nerve	16	0	0	0	0	....
Injection of alcohol into gasserian ganglion	4	0	0	1	1	25.0
Subtotal rhizotomy (2d and 3d branches)	466	15	12	11	38	8.2
Subtotal rhizotomy (1st branch)	32	4	4	0	8	25.0
Total rhizotomy (all branches)	360	22	69	20	111	30.8
Totals	878	41	85	32	158	18.0
Incidence, percentage		4.7	9.7	3.6		

Incidence of Corneal Lesions.—The corneal lesions which followed these operations for trigeminal neuralgia are summarized in table 1. The incidence of the corneal lesions occurring for each type of operation and the incidence for all operations are indicated. The following conclusions can be drawn:

1. Injections of alcohol into the peripheral branches are without significance in the production of corneal lesions.

2. Injection of alcohol into the gasserian ganglion, if one may draw conclusions from so few cases, is no less dangerous than section of the sensory root of the ophthalmic division of the trigeminal nerve.

3. Subtotal rhizotomy involving the second and third branches resulted in corneal lesions in only 8 per cent of cases.

4. Subtotal rhizotomy involving the first, or ophthalmic, branch and total rhizotomy produced paralysis of the ophthalmic branch, resulting in the root fibers to the ophthalmic branch is done. Corneal anesthesia is not to be expected in cases in which an incomplete rhizotomy involving the second and third branches only is performed. Actually, of the 466 incomplete rhizotomies involving only the second and third branches, 82, or 18 per cent, did result in anesthesia of the cornea.

Lagophthalmos.—Paralysis of the facial nerve is a complication encountered in a small percentage of cases. It developed in 52 of the 878

TABLE 2.—*Incidence of Complicatoins in Operations for Trigeminal Neuralgia (University Hospital, 1927-1943)*

Complications	Number	No Complication	Corneal Anesthesia	Paralysis	Paralysis and Anesthesia	Number	Incidence, Percentage
Injection of alcohol into branch of fifth nerve	16	16	0	0	0	0	0
Injection of alcohol into gasserian ganglion	4	2	0	1	1	2	50.0
Subtotal rhizotomy (2d and 3d branches)	466	370	69	14	13	96	20.6
Subtotal rhizotomy (1st branch)	32	1	27	0	4	31	96.9
Total rhizotomy (all branches)	360	3	338	0	19	357	99.2
Totals	878	392	434	15	37	486	
Incidence, percentage	...	...	49.3	1.7	4.2	55.2	

corneal anesthesia. The incidence of corneal lesions, 25 and 30 per cent respectively, was relatively high.

This difference in the incidence of corneal lesions, 8 per cent for rhizotomies involving the maxillary and mandibular nerves and 25 to 30 per cent for operations involving the ophthalmic nerve, explains why neurosurgeons expressly try to leave the fibers to the first branch uncut if possible.

cases, or in 5.6 per cent. In 15 cases the paralysis was not associated with corneal anesthesia, and in 37 cases it was so associated.

OPERATIVE COMPLICATIONS IN RELATION TO INCIDENCE OF CORNEAL LESIONS

Table 3 shows the relation of corneal anesthesia, paralysis of the orbicularis muscle without anesthesia and paralysis of the orbicularis muscle with anesthesia to the incidence of corneal lesions.

TABLE 3.—*Relation of Operative Complications to Corneal Lesions Following Operations for Trigeminal Neuralgia (University Hospital, 1927-1943)*

		Corneal Lesions				
Complications	Number	Diffuse Epithelial Vesiculation	Neuroparalytic Keratitis	Exposure Keratitis	Number	Incidence, Percentage
No complication	392	0	0	1	1	0.25
Corneal anesthesia	434	55	73	0	128	29.5
Paralysis of orbicularis muscle	15	0	0	6	6	40.0
Corneal anesthesia and orbicular paralysis	37	0	12	12	24	64.9
Totals	486	55	85	18	158	
Incidence, percentage		11.3	17.5	3.7	32.7	

OPERATIVE COMPLICATIONS

Two complications may follow operations for trigeminal neuralgia which are important in relation to the occurrence of corneal lesions, namely, corneal anesthesia, due to paralysis of the ophthalmic branch of the fifth nerve, and lagophthalmos, due to paralysis of the seventh nerve.

Corneal Anesthesia.—Such a complication is to be expected in all cases in which a complete rhizotomy or an incomplete rhizotomy involving

Absence of Complications (no anesthesia or paralysis).—In 392 cases the operation for trigeminal neuralgia was not followed by either corneal anesthesia or paralysis of the orbicularis muscle. In these cases corneal lesion occurred in only 1 case. The lesion was diagnosed as "exposure keratitis" and was described as a corneal abrasion with linear staining. It responded readily to simple treatment. One can conclude from this series, therefore, that operation on the lower branches of the trigeminal

nerve which does not injure the first branch of the fifth nerve or the seventh nerve does not result in corneal damage.

Corneal Anesthesia.—Of 434 cases in which this complication occurred, including cases in which the root fibers to the first branch were severed and cases in which anesthesia occurred as a complication of operation on the lower branches, corneal lesions developed in 128, or 29.5 per cent. In 55, or 12.7 per cent of the total number of cases of corneal anesthesia, the lesion was mild—superficial epithelial vesiculation—and in 73, or 16.8 per cent, the condition was a severe form of neuroparalytic keratitis.

Paralysis of the Orbicularis Muscle.—This complication occurred unassociated with corneal anesthesia in 15 cases. In 6 cases, or 40 per cent of this group, exposure keratitis developed. In all these cases the lesion was severe enough to require tarsorrhaphy.

Paralysis of the orbicularis muscle in association with corneal anesthesia occurred in 37 cases. The seriousness of this combination is shown by the development of corneal lesions in 24, or 64.9 per cent, of the cases. In 12 of these cases exposure keratitis and in 12 cases neuroparalytic keratitis was present. External, or three band, tarsorrhaphy was the favored method of treatment in these cases.

When the series of cases is considered as a whole, corneal anesthesia or paralysis of the orbicularis muscle or corneal anesthesia plus paralysis of the orbicularis muscle occurred as a complication in 486 cases. Of this number, superficial epithelial vesiculation developed in 55, or 11.3 per cent; severe neuroparalytic keratitis in 85, or 17.5 per cent, and exposure keratitis in 18, or 3.7 per cent. Corneal lesions occurred in a total of 158 cases, an incidence of 32.7 per cent.

The relative seriousness of the various lesions is indicated by the final visual acuity. An attempt was made through correspondence to determine the visual acuity of all patients who

TABLE 4.—*Types of Operations for Trigeminal Neuralgia in Relation to Corneal Lesions (University Hospital, 1927-1943)*

Operation	Number	Anesthesia	Paralysis	Diffuse Epithelial Vesiculation	Neuroparalytic Keratitis	Exposure Keratitis	Incidence: Number	Incidence: Percentage
A. Repeated operations								
First operation								
Subtotal rhizotomy (2d and 3d branches)	5	0	1	0	0	1	1	20.0
Subtotal rhizotomy (1st and 2d branches)	3	3	0	1	0	0	1	33.3
Second operation (total rhizotomy)	8	8	1	1	2	0	3	47.5
Incidence, percentage (second operation)	..	..	..	12.5	25.0		37.5	
B. Operations complicated by neoplasm								
Carcinoma of face, sinuses or mouth						13		
Tumor involving gasserian ganglion						8		
Other intracranial tumors						3		
Carcinoma of breast						1		
Intractable pain in left eye (cause undetermined)						1		
Total						26		

Operation	Number	Anesthesia	Paralysis	Diffuse Epithelial Vesiculation	Neuroparalytic Keratitis	Exposure Keratitis	Incidence: Number	Incidence: Percentage
Subtotal rhizotomy (2d and 3d branches)	2	0	0	0	0	0	0	....
Total rhizotomy	24	24	7	1	4	3	8	33.3
Totals	26	24	7	1	4	3	8	
Incidence, percentage			26.9	3.8	15.5	11.5	30.8	
C. Bilateral operations (not total rhizotomies on both sides)								
Both operations subtotal (2d and 3d branches)								
First operation	3	0	0	0	0	0	0	0
Second operation	3	0	0	0	0	0	0	0
Subtotal and total rhizotomies								
First rhizotomy subtotal (2d and 3d branches)	1	0	0	0	0	0	0	0
Second rhizotomy total or subtotal (1st branch)	1	1	0	0	0	0	0	0
First rhizotomy total or subtotal (1st branch)	4	4	0	1	0	0	1	25
Second rhizotomy subtotal (2d and 3d branches)	4	0	0	0	0	0	0	0
Totals	16			1			1	
Incidence, percentage				6.2			6.2	
D. Bilateral operations (total rhizotomies on both sides)								
Both rhizotomies total or subtotal (1st branch)								
First operation	6	6	0	1	3	0	4	....
Second operation	6	6	0	1	0	0	1	....
Totals	12			2	3		5	
Incidence, percentage				16.7	25		41.7	

had corneal anesthesia. About one half replied, with the following data.

Corneal Lesion	No. of Patients
Diffuse epithelial vesiculation.....	55
33 replied, of whom 27 had reading vision	
Severe neuroparalytic keratitis.....	85
33 replied, of whom 11 had reading vision	
Exposure keratitis	18
7 replied, of whom 5 had reading vision	

SPECIAL GROUPS OF CASES IN RELATION TO INCIDENCE OF CORNEAL LESIONS

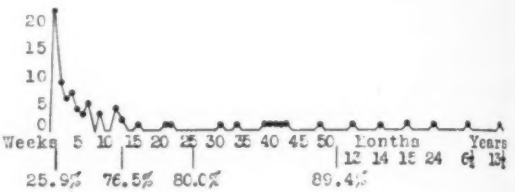
Three groups of cases are of interest from the standpoint of their relation to corneal lesions: namely cases with repeated operations, those complicated by neoplasm and those cases with bilateral operations.

Repeated Operations (table 4 A).—In 8 cases a second operation was done on the same side on which a previous operation had been performed. In 2 of these a mild corneal lesion followed the first operation. In both cases severe neuroparalytic keratitis followed the second operation. This observation is of interest in that the lesion following the first operation may indicate that a lesion is more likely to follow the second operation.

Operations Complicated by Neoplasm (table 4 B).—In 26 cases pain in the distribution of the trigeminal nerve was associated with a neoplasm involving the branches or the ganglion of the fifth nerve, either by direct extension or by metastasis. This was recognized before operation in most cases, and rhizotomy was performed for "intractable pain." In this group it is interesting to note that there was a higher incidence of cases of paralysis of the seventh nerve, 26.9 per cent as compared with 6 per cent in cases of uncomplicated rhizotomy. The total incidence of corneal lesions was not higher, 30.8 per cent, but the incidence of exposure keratitis was greater, 11.5 per cent, as compared with 3.6 per cent for the cases of uncomplicated rhizotomy.

Bilateral Operations.—In 14 cases rhizotomy was performed on both sides. This group is divided into two parts: 1. Eight cases in which a complete rhizotomy was not done on both sides (table 4 C). There occurred among these cases only 1 instance of corneal lesion—diffuse epithelial vesiculation. The incidence of 6.2 per cent is not greater than the incidence for single operations.

2. Six cases (12 operations) in which a complete rhizotomy was done on both sides (table 4 D). Parsons [2] stated that "neuroparalytic keratitis occurs in all cases of paralysis of both fifth nerves." The results in the present series, however, do not confirm this statement. Of this series of 6 cases, with 12 operations, a mild corneal lesion developed in both eyes in 1 case seven years after the operation. Severe neuroparalytic keratitis occurred after the second operation in 2 cases. In both cases it developed in the eye that had been operated on first, but after the second operation. In both cases there was satisfactory vision in the other eye over a year later. Here, the relation of the second operation is a little obscure; it would be expected that the eye operated on most recently would be more susceptible to neurotrophic disturbance.



Time of onset of neuroparalytic keratitis in 85 cases in which an operation was performed for trigeminal neuralgia. The abscissa indicates the number of weeks (months or years) after operation at which the lesion appeared; the ordinate, the number of cases.

The condition developed in the hospital in 22 cases, or 25.9 per cent; within one month in 44 cases, or 51.8 per cent; within three months in 65 cases, or 76.5 per cent; within six months in 68 cases, or 80 per cent, and within one year in 76 cases, or 89.4 per cent.

TIME OF ONSET OF NEUROPARALYTIC KERATITIS

A study of the time of onset of neuroparalytic keratitis revealed certain interesting facts.

1. In only about 25 per cent of cases did the lesion develop in the hospital, that is, within the first week.

2. In the other 75 per cent of cases the condition developed after the patient went home, when he presumably was in a less favorable position to have prompt, efficient treatment.

3. In 80 per cent of cases the onset was within six months.

These observations are important with relation to treatment. They indicate that the first six months is the period in which the eye is most susceptible to neurotrophic disturbance. After this period, presumably, the patient is able to readjust his cellular metabolism and becomes less susceptible. Some danger, however, always exists.

2. Parsons, J. B.: Diseases of the Eye, London, J. & A. Churchill, Ltd., 1934, p. 222.

TREATMENT

Treatment of this condition has varied somewhat over the seventeen year period of this study, but the importance of closure of the lids has always been recognized. A mild antiseptic, such as an aqueous solution of metaphen, 1:2,500, or of merthiolate, 1 : 5,000, is prescribed. The stronger antiseptics, silver and zinc, are not now used, and cautery with trichloroacetic acid is avoided. Isotonic solution of three chlorides U. S. P. is used frequently during the day if there is an indication of insufficient secretion of tears. Closure of the lids has been obtained by various means, such as padding the eye, closing with adhesive tape, use of the Frost suture and tarsorrhaphy. A Buller shield, to prevent desiccation, was frequently employed. The use of eye pads has in general been discontinued because of the danger of their scratching the cornea if the lids are open. If pads are used, it is important to apply plenty of petrolatum to them. Closing the lids with adhesive tape is a better procedure, but the tapes may loosen and allow the eye to open and become traumatized. A Frost suture is the best procedure for temporary closing and will hold for from a week to ten days. The device is useful in cases in which the patient is uncooperative or comatose.

A tarsorrhaphy is the best procedure for treatment of neuroparalytic keratitis or exposure keratitis—either a three band or an Elschnig lateral tarsorrhaphy, as indicated. The tarsorrhaphy should be left in place for six months.

Cautery of the punctum was employed in a limited number of cases; it is useful in cases in which there is a notable decrease in secretion of tears. Cervical sympathectomy has also been advised. It was used in only a few cases in this series.

The best treatment emphasizes prophylaxis. Prophylactic care should be used with any eye which is anesthetic. Temporal shields may be applied to the glasses, and isotonic solution of three chlorides U. S. P. should be used several times daily if the test for lacrimal secretion shows a deficiency of tears. Early tarsorrhaphy should be performed in doubtful or suspected cases. If the tarsorrhaphy is left in place for six months, the patient will have been carried through the most critical period in the development of this disease. One should remember that only in 25 per cent of the cases does the lesion develop in the hospital. In 25 per cent it develops during the first month at home, and in another 25 per cent during the next two months. These are the cases that cause embarrassment to the neurosurgeon and the ophthalmologist.

CONCLUSIONS

1. Operations on the lower branches of the trigeminal nerve which do not involve the first, or ophthalmic, branch do not cause damage to the cornea.

2. Corneal anesthesia and lagophthalmos are the important factors in the production of corneal lesions.

3. Combined corneal anesthesia and lagophthalmos give the greatest incidence of corneal lesions (65 per cent).

4. With repeated operations, a lesion occurring after the first operation may indicate that a lesion is likely to follow the second operation. But the incidence of lesions was not definitely greater with repeated operations than with single operations.

5. Operations complicated by the presence of neoplasm are more likely to be followed by paralysis of the seventh nerve and exposure keratitis. But the incidence of corneal lesions was not greater than with other complete rhizotomies.

6. The prospect of development of neuroparalytic keratitis after complete rhizotomy on both sides is not greater than that after a complete rhizotomy on one side and should not deter one from bilateral operation if severe pain is present.

7. The patient with corneal anesthesia following operation for trigeminal neuralgia is most susceptible to development of neuroparalytic keratitis during the first few months immediately after operation.

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ABSTRACT OF DISCUSSION

DR. A. D. RUEDEMANN, Cleveland: Dr. Pannabecker's comprehensive report should not lead one astray as to the seriousness of the complications of neuroparalytic disease of the cornea. I have had my saddest experiences with this condition.

In the treatment of trigeminal neuralgia the surgeon should not cut the first division of the nerve. Unfortunately, the ophthalmologist does not see the patient until that operation has been performed. Then it is often too late for the oculist to get the best visual result. On his leaving the hospital, the patient should be referred to an oculist near home. The patient is not always satisfied with the exchange of anesthesia of the face for constant observation of the eye and is inclined to go to the oculist nearer home first. Recently a patient consulted me after the development of a severe perforating ulcer. The patient usually has no discomfort;

and unless some one has the eye under observation, the condition may progress beyond repair.

I recall a patient who was sent to me from a neighboring city. After operation a severe corneal ulcer developed. Under treatment the cornea healed, but the resulting scar, which, as is usually the case, was below the pupil, distressed the patient. She sued me because she said the scar was due to my treatment, which had been use of atropine and application of heat by the nurse at the office. The patient said that the heat was applied at too high a temperature and caused secondary scarring. I wish to point this out because the patient does not always appreciate relief of the pain. Ocular complications are unpleasant in that the scarring is below the pupil. If this unfortunate sequela of neuroparalytic keratitis develops in addition to the facial paralysis, the eye may be lost.

My last patient with this complication had a corneal ulcer. After sewing the lids together and using all known methods, I applied a conjunctival flap, which did not maintain the position better than any other procedure. Finally, because of perforating ulcer, the eye had to be enucleated. In order to obtain a good cosmetic result, I planted the lucite ball within the socket, without benefit.

Accordingly, the best treatment is prevention of the process on the cornea, unless the neurosurgeon can be persuaded to do a less radical procedure in the first place and thus prevent the complication of neuroparalytic keratitis.

Therapy at home must be rigid. The patient must be observed daily by some member of the family. His general health must be maintained. If an ulcer of the eye should develop in spite of constant observation, a scar is most likely to form below the pupil, which causes serious diminution in vision.

With neuroparalytic keratitis there is decreased tearing, as well as diminution in the secretion of the meibomian glands and the glands which oil the cornea. It is this desiccation that precedes the desquamation of the corneal epithelium, with secondary invasion by organisms, which finally causes scarring and subsequent difficulty.

Patients should be referred to a local physician after operation for trigeminal neuralgia. It will relieve the ophthalmologist of the responsibility, and the condition of the eye can be more satisfactorily watched.

MAJOR J. M. MASTERS, Medical Corps, Army of the United States: It is difficult to add anything significant to a paper as complete as Dr. Pannabecker's. His critical study of an unusually large number of patients with trigeminal neuralgia treated by different surgical methods has provided many substantial facts. Statistical information such as his lifts into the realm of certainty the impressions and clinical conceptions which many ophthalmologists get from seeing relatively few patients.

About ten years ago I had the opportunity of studying the eyes of about 15 patients who were treated by either total or subtotal posterior ganglionectomy or rhizotomy, in the neurosurgical service of Dr. E. Vernon Hahn, of Indianapolis. My impressions gained from these patients were subsequently confirmed by the study of other patients similary treated.

Loss of luster of the corneal epithelium soon after operation, with the appearance of a faint gray cast, was nearly always observed, causing apprehension of a catastrophe which did not always occur. Its recognition is probably not original with me, as it corresponds, in time of onset at least, with the "trophic pitting of the cornea" which was found in 1892 by Gaule to occur in the first few hours after section of the gasserian ganglion. No doubt many ophthalmologists have seen the same thing. It is the simplest and most benign form of neurotrophic disturbance of the cornea and corresponds to the "central edema" of the corneal epithelium, characterized by punctate stippling.

From one to three days after operation a fine stippling of the involved cornea may be observed after staining with fluorescein. This stippling is seen much earlier than the more serious corneal complications mentioned by Dr. Pannabecker. It is as though the end of a very fine hair had been dipped in green dye and then dotted lightly over the corneal surface. Tiny green pinpoints may be either scattered over the entire surface or limited to smaller areas between the margins of the lids. Fine straight or angular lines or, less frequently, dull-staining spots of confluence may lie between them. These points may be seen more easily on some days than on others. Their presence indicates cellular edema and fine exfoliation of the epithelium. Usually they leave five to seven days after operation without secondary scarring, but they have been observed to last many weeks without going on to ulceration. Frequent relapses may occur during the process of healing. Greater sloughing and formation of vesicles exceed them in degree and are not an essential part of the entity. They indicate an advanced pathologic process necessitating tarsorrhaphy. Uveal inflammation is not a counterpart.

The observation has never been made in cases of subtotal rhizotomy in which the ophthalmic division of the nerve has been spared. On the other hand, the corneas in 2 cases which had been rendered anesthetic exhibited no change. An identical lesion of the cornea is sometimes seen in cases of herpes zoster; in these cases healing is often delayed.

Although the manifest pathologic process is slight, it seems to gain importance from its very

insignificance. Its recognition will be reassuring to the apprehensive patient who has discovered dimness of vision on the side corresponding to his recent operation, or on the side of the "shingles," and to the oculist who has discovered it for himself. This does not justify a relaxation of his vigilant anticipation of more serious trouble which may follow or in any way minimize the importance of early suture of the lids in cases in which formation of ulcer is imminent. However, the realization that there is such an entity which will of itself run a benign course may be valuable information for the surgeon.

Dr. C. L. Pannabecker, Ann Arbor, Mich.: I did not have time to say all I should like to have said on the treatment of the disease, but I heartily agree with Dr. Ruedeman's comments. The condition is a serious one and needs to be carefully watched. I thank Dr. Masters for his suggestion with regard to the minor lesions, the early lesions, which are frequently encountered. They also indicate that the cornea is susceptible to damage and should be watched carefully. If it is impossible to follow such a lesion, one is justified in doing a tarsorrhaphy. In any case, if the lesion is suspicious tarsorrhaphy is indicated.

BLOOD VESSELS OF THE CONJUNCTIVA

STUDIES WITH HIGH SPEED MACROPHOTOGRAPHY

SAMUEL GARTNER, M.D.

NEW YORK

A new technic of high speed photography produces magnified pictures of an excellent character and offers an additional method for study of the eye. The blood vessels of the conjunctiva have been found to be unusually available for photography because they lie in an almost transparent medium, and the red vessels afford good contrast to the background of the opaque white sclera.

I have photographed the eye with many types of cameras but have found drawbacks with all of them. The intensity of light is limited by the tolerance of the human eye, and that is greatly diminished with disease. As the eyeball is curved, it is difficult to get a large section of it in clear focus on a flat film. Even in a cooperative patient there is constant movement of the eye, which blurs the picture. High speed photography overcomes many of these difficulties.

THE CAMERA

The essential improvement has been in the light source, which is a bulb filled with xenon, krypton and argon gases, developed by Edgerton.[1] This light flashes for approximately one-fifteen-thousandth second, with an intensity of about 2,000,000 international candles. A power pack somewhat similar to a type used in radio is employed. It operates on a 110 volt alternating current, which is raised to 2,000 volts, rectified and stored in condensers. The energy is discharged through a special tube in one-fifteen-thousandth second. With this light pictures have been taken of rapidly moving objects, and they appear to be standing still. For instance, when the revolving blades of an electric fan are photographed, no motion can be detected. The light is balanced for color photography.

Pearce[2] developed an ingenious camera for ophthalmic photography into which this light was incorporated. It was employed in a study of the limbal capillaries in cases of riboflavin deficiency. Further improvements were made by Fairchild. A focusing light is attached to a fine single lens reflex camera. The viewing and focusing are done on a ground glass. When the switch is thrown, the focusing light goes out automatically; the diaphragm stops down to a predetermined opening between f 18 and f 120; the bulb flashes, and the picture is taken. The illustrations in this article were photographed on orthochromatic film with the diaphragmatic opening set between f 80 and f 100. A head rest of the type usually employed with the biomicroscope is provided. The camera is mounted on a movable stage for ease and accuracy in focusing.

The photographs in this study were taken at a magnification of 3.5 and were enlarged as much as ten times, to a magnification of 35. They were not retouched. With other lenses pictures at higher magnifications are possible. The range of magnification is similar to that commonly employed with the biomicroscope. I decided to call this method macrophotography, which means the taking of enlarged pictures, and to differentiate it from photomicrography, which signifies magnification of pictures taken through a microscope.

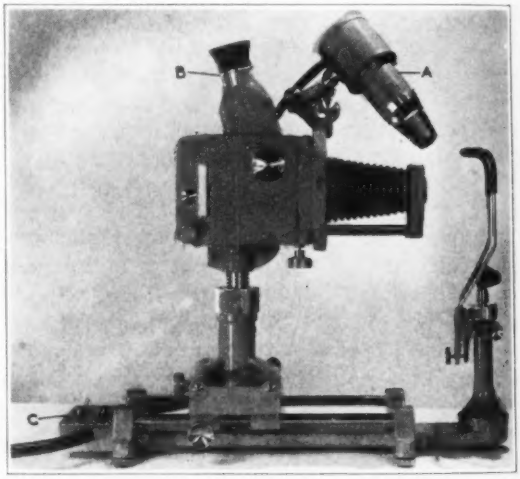

Fig. 1.—Camera, built by Fairchild Instrument and Camera Company, New York. *A*, projector, containing focusing and flash lights, adjustable to various positions; *B*, view finder; *C*, switch controlling a motor which automatically turns out the focusing light, sets the diaphragm at a predetermined opening, releases the flash and winds the film.

The patient's reaction to the light is neglible. There is awareness of a sudden bright flash, but its extremely short duration makes the light tolerable, without any apparent discomfort. There is no sense of heat from the flash. I exposed my eye for the taking of four pictures in a minute without being annoyed by the light, nor could I sense an after-image. Ordinary photographic bulbs emit much less light but the flash

From the Ophthalmological Service of Montefiore Hospital for Chronic Diseases.

1. Edgerton, H. E.; Germehausen, K. J., and Grier, H. E.: High Speed Photography, J. Roy. Photographic Soc. Great Britain **76**:198, 1936.

2. Pearce, cited by Tisdall, F. F.; McCreary, J. M., and Pearce, H.: The Effect of Riboflavin on Corneal Vascularization and Symptoms of Eye Fatigue in R.C.A.F. Personnel, Canad. M. A. J. **49**:5, 1943.

lasts from one-fifteenth to one-two-hundredth second and usually produces an after-image. The effect on the eye seems to depend on the intensity of the light and its duration. Further studies on the effects of this illumination on the retina are planned. The magnified pictures show details of the human eye that it has never before been possible to photograph. The minute structure of the conjunctiva and its vessels are well demonstrated. The cornea, the iris and the visible portion of the lens are available for study with this technic. It is interesting to compare these pictures with drawings in textbooks and then to realize the difficulties of observation and faithful sketching.

scope were reported on by Koeppe,[4] Ruedemann[5] and others. In this study I checked many of the photographs by examination with the biomicroscope.

The blood vessels are in two layers—a deep layer on the episclera and a superficial one just beneath the mucosa, with anastomoses between them. The anterior ciliary arteries are in the deep layer and travel around the eyeball on the rectus muscles, on each of which there are

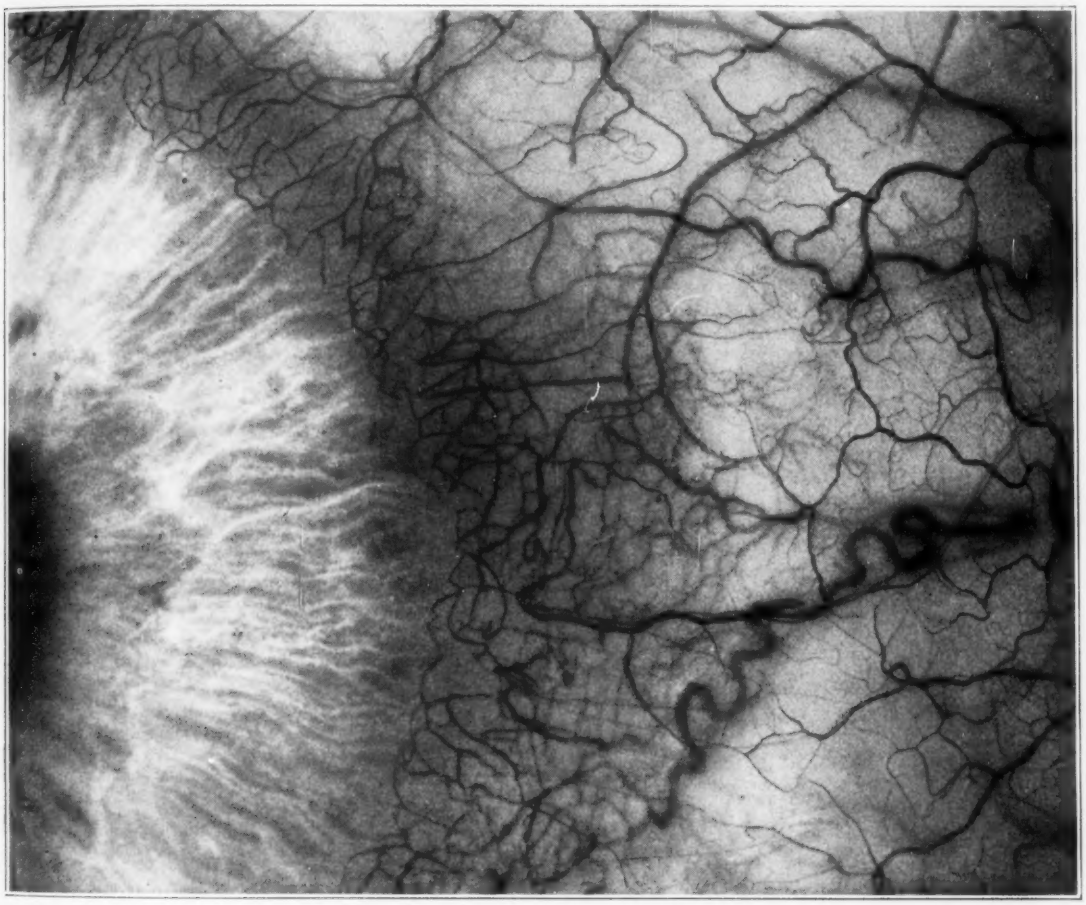

Fig. 2.—Normal capillaries at the limbus of a man aged 27; × 14.

THE CONJUNCTIVA

The white of the eye is far from white, but it is an almost transparent tissue, with a rich vascular network overlying the opaque sclera. This transparency may be interfered with, especially in older persons, by spots of pigment, pinguecla or other blemishes. Edema may interfere with the clarity of a picture. It seems to me that of all the exposed parts of the body the conjunctiva reacts most readily in the formation of edema, and minimal trauma will induce it.

Leber[3] described the conjunctival vessels in great detail. Observations with the biomicro-

usually two arteries, except for the external rectus muscle, which usually carries one, and occasionally no visible, artery. About 3 or 4 mm. from the limbus these arteries divide into two branches. The larger branch penetrates the

3. Leber, T.: Die Zirculations und Ernährungsverhältniss des Auges, in Graefe, A., and Saemisch, T.: Handbuch des gesamten Augenheilkunde, ed. 2, Leipzig, Wilhelm Engelmann, 1903, vol. 2, pt. 1.

4. Koeppe: Intravital Microscopy of Eye with Gullstrand-Nernst Lamp, Deutsche med. Wchnschr. **46**:630, 1920.

5. Ruedemann, A. D.: Conjunctival Vessels, Tr. Sect. Ophth., A. M. A., 1933, p. 114.

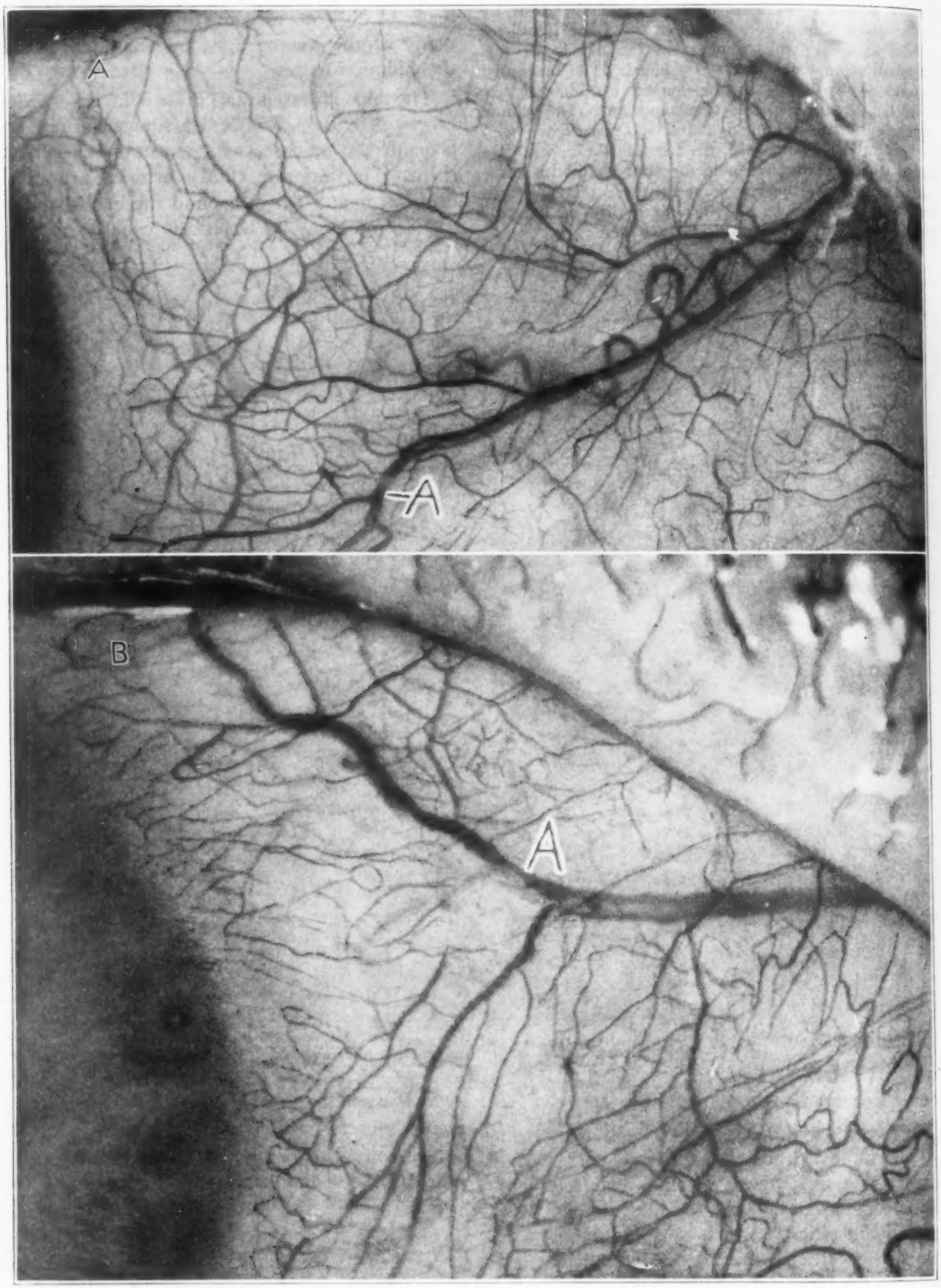

Fig. 3.—(*a*) Aqueous vein (*A*) in a woman aged 41. A paler vessel with a high content of aqueous joins the large conjunctival vein. At first there is layering of blood and aqueous in the vein, and later the streams merge. × 14.5. (*b*) Aqueous vein (*A*) in a female aged 70. A paler vessel, with a high aqueous content, joins another conjunctival vein before entering the large vein. The layering at the beginning is evident, and later merging of aqueous and blood is noted. × 18.

globe to anastomose with the long posterior ciliary arteries. The smaller branch supplies the conjunctiva and often anastomoses with branches from the other ciliary arteries to form a broken or irregular circle about the limbus. The anterior ciliary arteries and their branches are characterized by pronounced tortuosity and are usually the most prominent of the conjunctival vessels. Smaller branches are obtained from the tarsal arteries at the outer ends of the fornices. They follow a straighter course and are more difficult to identify.

The conjunctival vessels are peculiar in the number that follow a parallel path. Occasionally these veins cross and then continue again in parallel formation. They should not be confused with aqueous veins, which will be discussed later. Some of the superficial vessels form bizarre patterns by a twisting and mazelike course. Small conjunctival hemorrhages were noted in a few of the cases of hypertension and diabetes, as described by Zeller.[6]

In childhood the conjunctival vessels are more delicate, with a regular and even caliber, and the

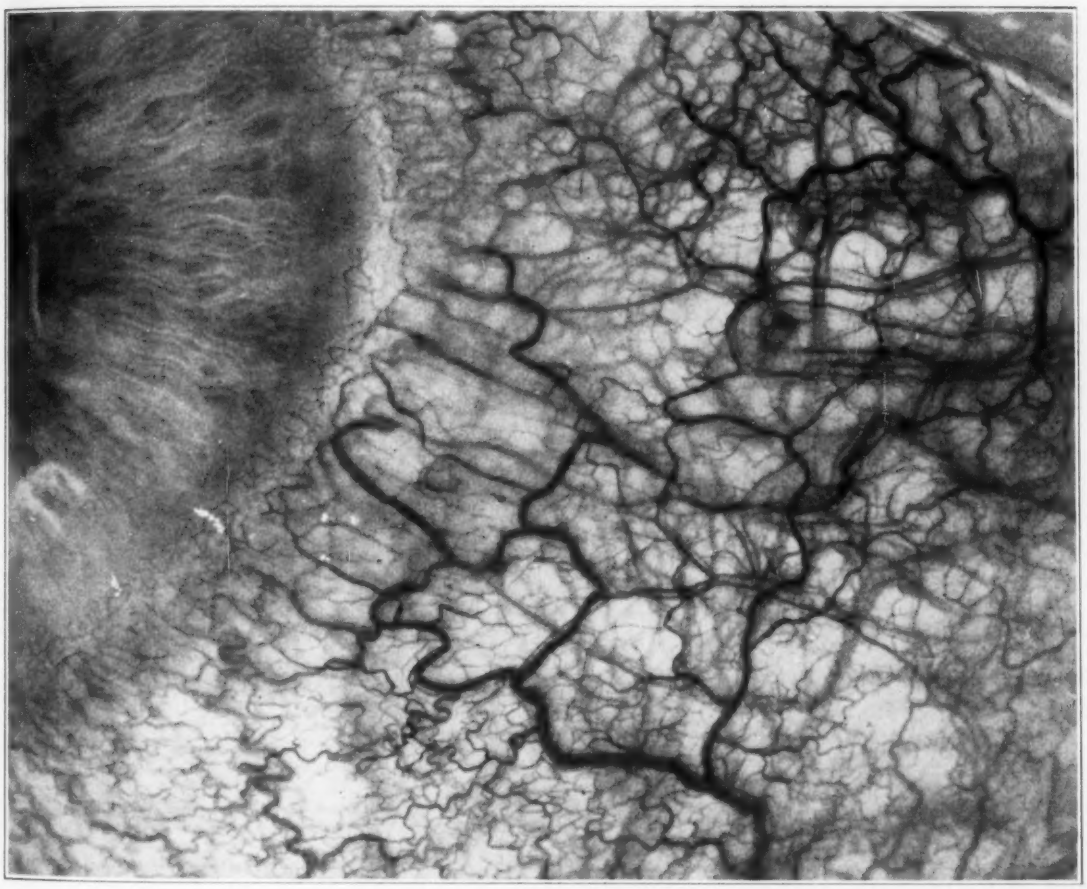

Fig. 4.—Acute conjunctivitis in a man aged 31. The superficial veins are notably dilated. The limbal capillaries are conspicuous. × 16.

The venous drainage is by a network of extensive ramification, chiefly in the superficial layer of the conjunctiva. The larger veins are in the horizontal meridian in the midcorneal section. It is often difficult to decide which of the smaller vessels are veins and which are arteries.

The most extensive network of capillaries in the conjunctiva is in the superficial layers at the limbus. From the limbus larger vessels radiate toward the fornix. In the midscleral zone many vessels follow a straighter course. Near the fornix the vessels are more irregular. anterior ciliary arteries are barely visible. After the age of 12 years the arteries become more tortuous and much more pronounced, and small irregularities often occur. The development of the vasculature of the conjunctiva is influenced by attacks of uveitis and conjunctivitis, which increase the number and size of the visible vessels, as well as their anastomoses. Irregular varicosities are often seen, especially in old and

6. Zeller, K.: Studien am Bindehautgefässen, Klin. Monatsbl. f. Augenh. **66**:609. 1921.

sclerotic persons and after ocular inflammation. After middle life, the veins are more prominent, and large, tortuous arteries with extensive anastomoses are common.

The capillaries are of particular interest because they form the largest part of the circulatory system and the area of greatest physiologic activity. In recent times interest has been renewed, as a result of a number of brilliant studies, particularly those of Krogh [7] and Lewis.[8]

The combined length of the capillaries of the body is almost astronomic, but only a few are readily available for study in man during life. In the human being extensive researches have been made on the capillaries in the nail bed, and to a lesser extent on the capillaries in other parts of the skin and on the tongue. In animals the capillaries in such areas as the webs of the toes, the ears and the mesentery have been studied. All of these methods have distinct limitations. The capillaries of the nail bed are in much less transparent tissue than the capillaries in the conjunctiva. Macrophotography of the conjunctival capillaries should enable workers to carry out their researches on capillaries in a better medium. The lymphatic supply in the conjunctiva is rich and can also be studied by this method. Of course, proper precautions are essential to respect the integrity of the eyeball.

The conjunctival capillaries extend to the limbus and are visible in the transparent sclera at the corneal border. An extensive literature has developed concerning the effects of riboflavin deficiency on the limbal capillaries. Bessey and Wolbach [9] and others demonstrated increased vascularity in the cornea of the rat with ariboflavinosis. This observation was applied by Kruse [10] and Sydenstricker [11] and their associ-

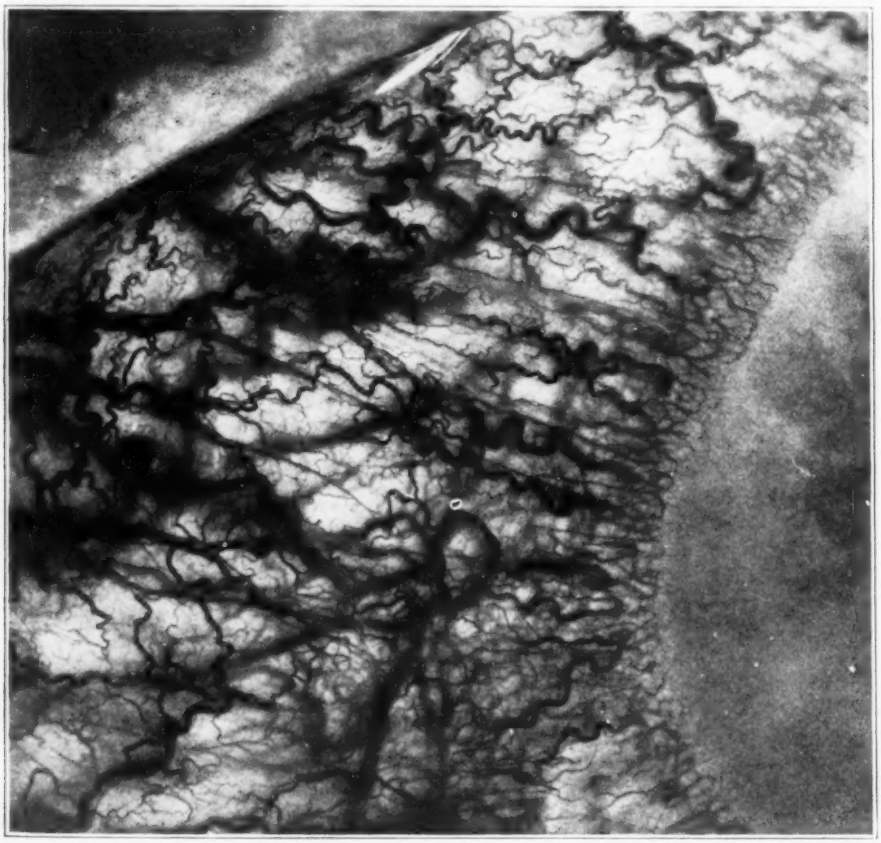

Fig. 5.—Acute uveitis and secondary glaucoma in a man aged 45, showing intense engorgement of the deeper set of arteries and the limbal capillaries. × 13.

7. Krogh, A.: The Anatomy and Physiology of Capillaries, New Haven, Conn., Yale University Press, 1930.

8. Lewis, T.: The Blood Vessels of the Human Skin and Their Responses, London, Shaw & Sons, Ltd., 1927.

9. Bessey, O. A., and Wolbach, S. B.: Vascularization of the Cornea of the Rat in Riboflavin Deficiency with the Note on Corneal Vascularization in Vitamin A Deficiency, J. Exper. Med. **69**:1, 1939.

10. Kruse, H. D.; Sydenstricker, V. P.; Sebrell; W. H., and Cleckley, H. M.: Ocular Manifestations of Ariboflavinosis, Pub. Health Rep. **55**:157, 1940.

(Footnotes continued on p. 7.)

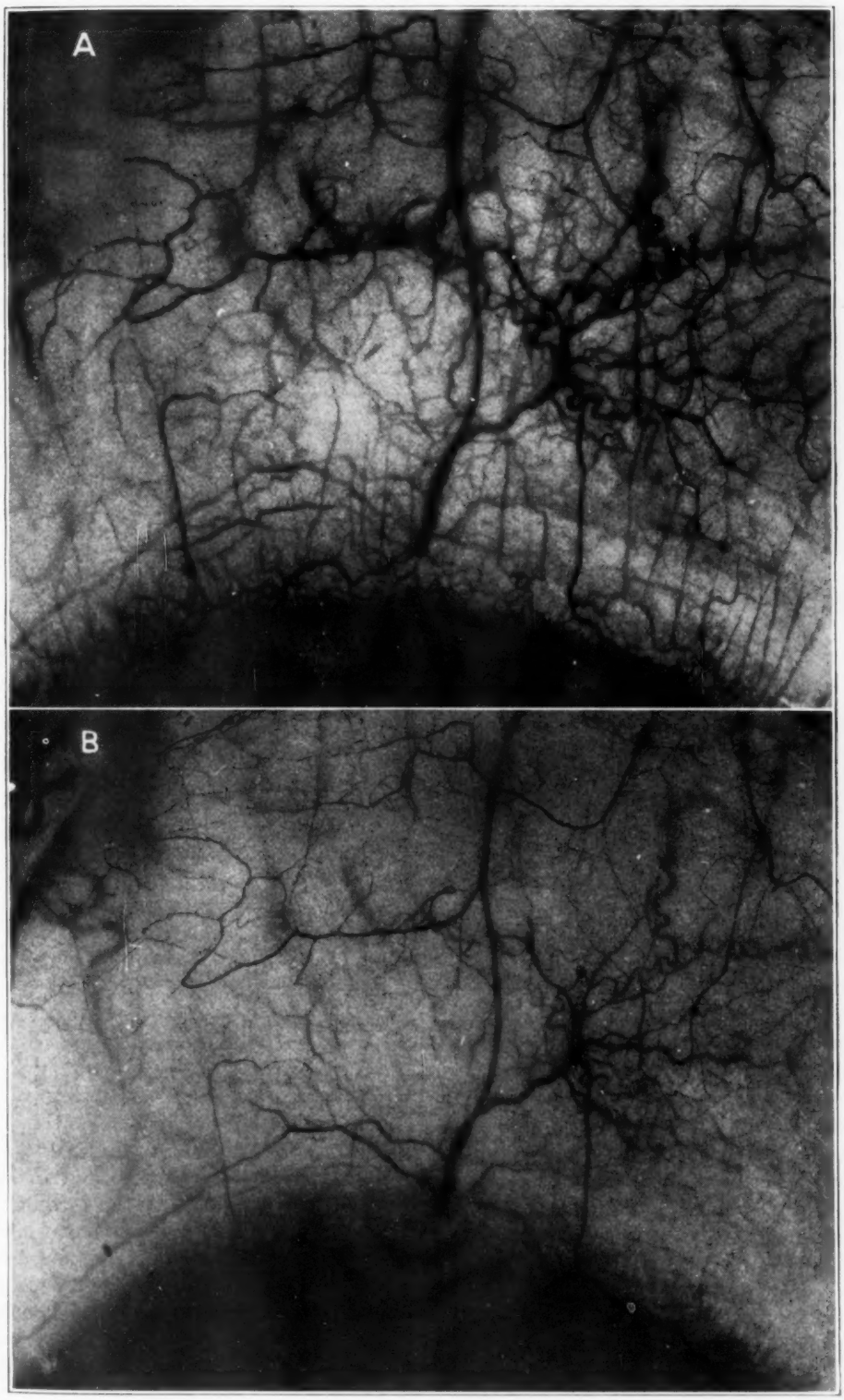

Fig. 6.—Reaction of the conjunctival vessels to epinephrine hydrochloride in a man aged 31 with hypertension. *A*, before medication; *B*, two minutes after instillation of 1 drop of epinephrine hydrochloride (1 : 1,000) in the conjunctiva, showing notable contraction of the veins. × 12.5.

ates as a diagnostic test in man for riboflavin deficiency. Recently, Lee and Hart,[12] in repeating the original studies on rats, found that severe keratitis, often with ulceration, caused the corneal vascularization that followed riboflavin deficiency. In man such keratitis has not been demonstrated. Vail and Ascher[13] and Pett,[14] among others, raised the question whether corneal vascularization furnishes a reliable guide to riboflavin deficiency. With the aid of photography evidence should be accumulated to settle this point.

ciation with conjunctivitis, uveitis and local irritation. It is questionable whether the collapsed capillaries are entirely empty, or whether they have a small lumen which contains plasma but is too small to admit red blood cells.

Inspection of the limbal capillaries through the biomicroscope has been the method used in diagnosis of ariboflavinosis, but I question whether this alone yields proper evidence. The degree to which the limbal capillaries are filled varies for each person from time to time; so comparisons

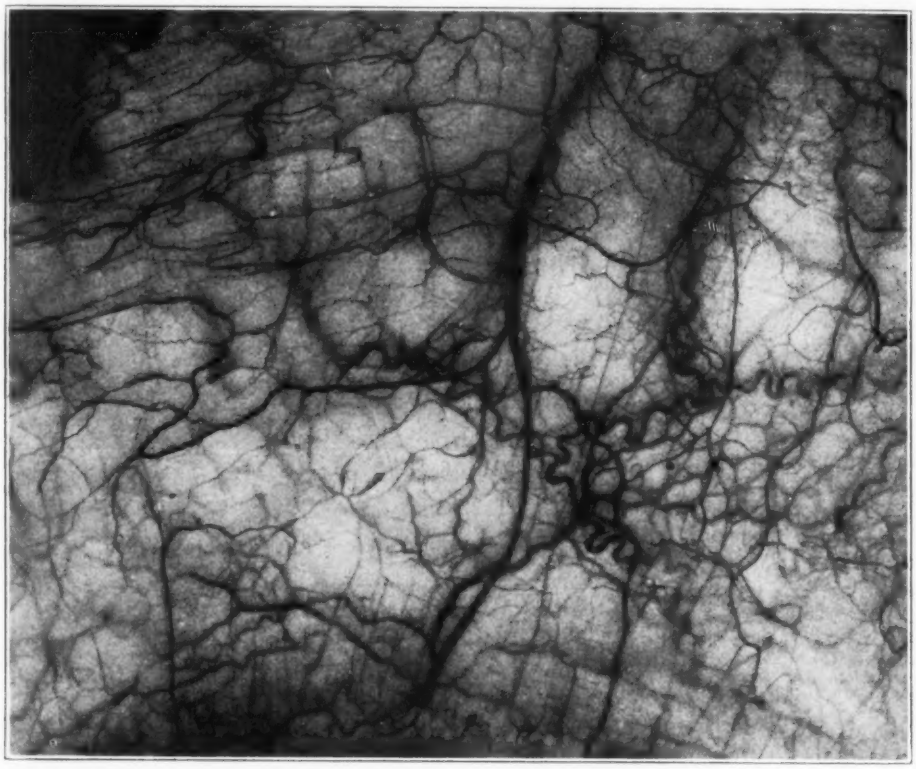

Fig. 7.—Photograph of the same area of the conjunctiva as that shown in figure 6, taken thirty minutes after instillation of the solution of epinephrine, when the vasodilation was greater than before the instillation. × 12.5.

Like capillaries all over the body, many of these vessels in the conjunctiva are collapsed most of the time and are invisible. With a proper stimulus, they react by opening the lumen, admit blood and become visible. This change should not be mistaken for a growth of new capillaries. The rapid filling of these vessels is noted in asso-

are not fair unless the conditions are controlled. For a critical survey the capillaries should be dilated to the maximum by a drug, such as histamine. This causes rapid and striking vasodilation, with notable increase in the number of visible capillaries, as will be shown later. After any form of therapy or deprivation, these capillaries should again be studied by the same method. Then it would be possible to decide whether there was really a loss or a gain in the number of capillaries.

AQUEOUS VEINS

Special adaptations of the ocular circulation provide for the drainage of aqueous. Schlemm's canal begins as a blind end which is arranged

11. Sydenstricker, V. P.; Sebrell, W. H.; Cleckley, H. M., and Kruse, H. D.: Ocular Manifestations of Ariboflavinosis, J. A. M. A. **114**:2437 (June 22) 1940.

12. Lee, O. S., and Hart, W. M.: The Metabolism of the Cornea, Am. J. Ophth. **27**:488, 1944.

13. Vail, D., and Ascher, K. W.: Corneal Vascularization Problems, Am. J. Ophth. **26**:1025, 1943.

14. Pett, L. B.: Influence of Riboflavin and Vitamin A on Eye Strain, Canad. M. A. J. **49**:293, 1943.

to receive the aqueous. Theobald [15] demonstrated that about twenty collectors drain Schlemm's canal into an intrascleral plexus, which, in turn, empties into the conjunctival veins. An important factor in the drainage of the aqueous is the intraocular pressure, which is about 25 mm. of mercury, while the pressure in Schlemm's canal is lower, the pressure in the intrascleral plexus is still lower and the pressure in the conjunctival veins, which is only 3 to 6 mm. of mercury is lowest of all. This pressure gradient explains the flow to a large extent. In the intrascleral plexus the aqueous and blood are usually well mixed, so that they reach the conjunctival vessels in an even stream. In many of the conjunctival veins the blood is rather pale, and there is evident separation of the blood cells, due to a high content of aqueous. Through these pale veins most of the conjunctival drainage of aqueous takes place.

In addition, there are other conjunctival veins in which the flow of aqueous is more clearly defined, since it is not well mixed with blood; Ascher [16] described these vessels in his excellent studies and called them "aqueous veins." In them may be seen a variety of conditions, ranging from clear aqueous to various mixtures of aqueous and blood. A peculiarity that makes these veins recognizable is a frequent layering of blood and aqueous in the vein.

The tributary with clear aqueous is not always seen, as it may enter from beneath the conjunctiva; and even when it is on the surface the vessel is often not well defined, as it lacks the coloring which defines blood vessels. When aqueous enters a vessel carrying blood, the two fluids do not immediately mix but travel along for some distance as separate layers and then gradually merge into an even stream. When tributaries carrying blood enter an aqueous vein, the blood cells can be seen to tumble into the clearer stream and set up eddies. The direction of flow is predominantly away from the limbus.

Ascher described aqueous veins after observing them with the biomicroscope. Many ophthalmologists have had difficulty in recognizing these veins; this method, therefore, should be an aid in locating them. Ascher reported that he observed aqueous veins in about 26 per cent of cases, while I found them in about 20 per cent. As noted by Ascher, these vessels are chiefly between the 1 and the 5 o'clock and between the 7 and the 11 o'clock positions. They can be detected from the midscleral zone to the limbus. Photographs show these vessels in a number of interesting variations.

A vascular phenomenon similar to aqueous veins was demonstrated by Hudack and McMaster [17] when dyes were seen to enter lymphatics. The stream of dye displaced and diluted another stream, itself unseen, which was derived from tissue remote from the region of injection, the two streams flowing unmixed for several millimeters.

Massage of the conjunctiva over an aqueous vein, or compression distal to the vessel, causes it to fill with a stream of red blood. Release of pressure is soon followed by layering of the stream. The entire aqueous vein is usually paler than the other vessels, as even the redder layer of blood is somewhat diluted with aqueous.

The pressure in the conjunctival veins is about 3 to 6 mm. of mercury. It is partly due to this low pressure that the aqueous and the blood do not rapidly mix together. There are a number of important differences in the physical and the chemical structure of aqueous and blood which govern the rate and extent of their mixture. According to Duke-Elder,[18] the solid content of the aqueous is 1.0869 per cent, while that of blood is 9.5362 per cent. The specific gravity of aqueous is about 1.008, while that of blood varies from 1.045 to 1.075. The surface tension of aqueous is greater than that of blood. Duke-Elder cited studies on the viscosity of the aqueous of the dog which showed that aqueous flowed through the Ostwald viscometer tubes in one minute and fifty-one and two-fifths seconds, while the flow of serum required two minutes and fifty-six and one-half seconds. As a result of these differences, the thorough mixing of blood and aqueous requires time and energy, with delay in their mixture in the aqueous vein.

The recognition of aqueous veins makes available for examination the vessels which carry a high concentration of aqueous. Further studies on these vessels is important, as they give information regarding the drainage of aqueous. The variations associated with glaucoma should be of particular interest.

The result of damage to the aqueous veins during an operation for glaucoma is important to determine, as such an effect might have con-

15. Theobald, G. D.: Schlemm's Canal, Tr. Am. Ophth. Soc. **32**:574, 1934.

16. Ascher, K. W.: The Aqueous Veins, Am. J. Ophth. **25**:1174 and 1301, 1942.

17. Hudack, S., and McMaster, P. D.: The Permeability of the Wall of the Lymphatic Capillary, J. Exper. Med. **56**:223, 1932.

18. Duke-Elder, W. S.: Text-Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1942, vol. 1, p. 435.

siderable influence on the intraocular tension. I have decided to examine my patients with glaucoma for the location of the aqueous veins before operation and, if possible, to avoid injury of these vessels. In cyclodiathermy, in which a wide area is treated, the results may be improved if the aqueous veins are not disturbed.

CONJUNCTIVITIS AND UVEITIS

In conjunctivitis the dilated superficial vessels are most prominent, while the deeper vessels are somewhat obscured by edema. In uveitis there is more obvious dilatation of the deeper arteries. In both conditions the limbal capillaries are engorged. Figures 4 and 5 demonstrate the vascular patterns.

DRUGS AND ALLERGENS

The conjunctival vessels react rapidly after any local stimulus, whether it is a foreign body, massage of the conjunctiva or instillation of a drug. The first reaction is similar with almost all local stimuli and consists of vasodilation and the filling of many capillaries which had been collapsed. Then comes the specific action of the drug or the stimulus used. The first reaction of vasodilation may be due to local nervous reflexes or to the production of histamine or the H-substance of Lewis.

A drug instilled in the conjunctival sac quickly diffuses through the epithelium and acts on the vessels. The diffusion through the entire conjunctiva is far from instantaneous, but is rapid from the point of application. A drug instilled in the lower cul-de-sac will affect the lower vessels first. For most extensive effects it is best to instil the medicament above the cornea. This method has the disadvantage that the cornea is more sensitive than the lower conjunctival fornix. Effects of instillation are readily

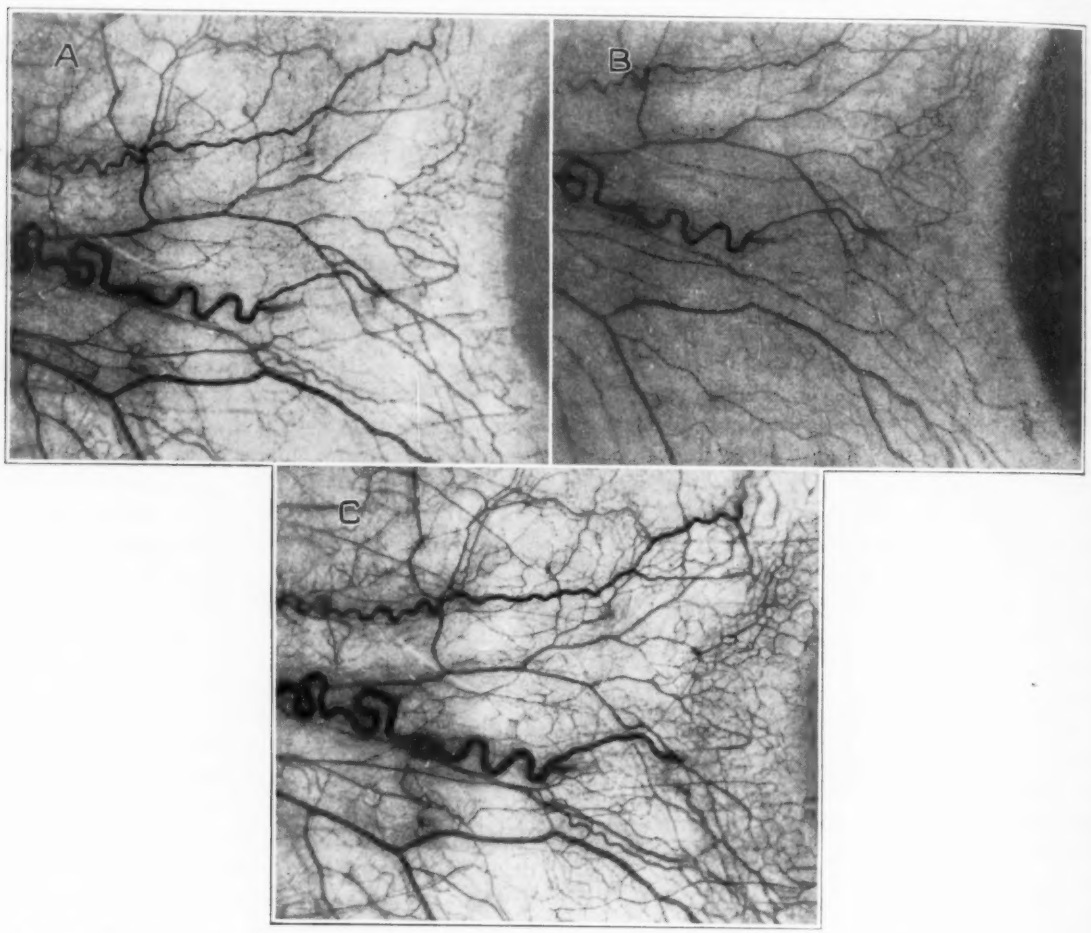

Fig. 8.—Reaction of the conjunctival vessels to physostigmine in a woman aged 70 with glaucoma.

A, before medication; *B*, twelve minutes after instillation of 1 drop of physostigmine salicylate, 0.5 per cent, into the conjunctival sac, showing notable contraction of the tortuous arteries, and *C*, fifty minutes after the instillation of physostigmine. There is vasodilation, especially of the arteries, and to some extent of the capillaries and veins. × 12.

observed in serial pictures of the conjunctival vessels, drugs with pronounced local effects, such as epinephrine or histamine, being used.

Epinephrine.—Epinephrine hydrochloride induces a well known arterial spasm. Lewis claimed that there is active capillary contraction as well. After instillation of 1 drop of the 1:1,000 solution there is a striking contraction of the arteries, followed by the disappearance of many capillaries and venules. The larger veins do not so readily become invisible. This effect reaches its maximum in about five minutes. Twenty minutes later there is a reaction in which a larger number of small vessels open than were visible before the instillation of epinephrine.

Curtailment of the arterial supply is the probable explanation for the temporary fall in intraocular tension produced by epinephrine in cases of glaucoma.

Physostigmine.—In 4 cases of glaucoma physostigmine salicylate, in 0.5 per cent solution, caused arterial constriction in five to fifteen minutes, followed by pronounced vasodilation, which was maintained for several hours.

Pilocarpine.—In 5 cases of glaucoma a 2 per cent solution of pilocarpine hydrochloride produced gradual vasodilation for about fifteen minutes, which then slowly receded to the previous level of contraction except that fewer capillaries were visible. It is interesting that there should be this difference in the vascular reaction to physostigmine and to pilocarpine.

An aqueous vein was followed in 1 of the cases in which pilocarpine was instilled. With the vasodilation there was a greater mixture of the aqueous with blood, which hid the column of aqueous. With the vasoconstriction which followed, there appeared a slight, but definite, increase in the layer of aqueous in this vessel.

Histamine.—Histamine biphosphate (1:50,000) caused rapid and striking vasodilation within fifteen seconds, which increased for about twenty minutes and then gradually diminished. Arteries, capillaries and veins shared in the dilation, which was accompanied by edema of the conjunctiva. Vasodilation with histamine is suggested as a method of making visible the limbal capillaries in studies on riboflavin.

Allergens.—The conjunctiva, like other mucous membranes and the skin, reacts abnormally and violently in allergic persons. The ocular test with tuberculin was described by Calmette in 1907. Allergic reactions of the eye were demonstrated in the early 1900's. In 1931 Peshkin [19] popularized the use of the conjunctiva for testing with dry pollen. He particularly pointed out that this membrane is more sensitive than the skin and that it often gave a positive reaction when the reaction of the skin was negative. In 1943

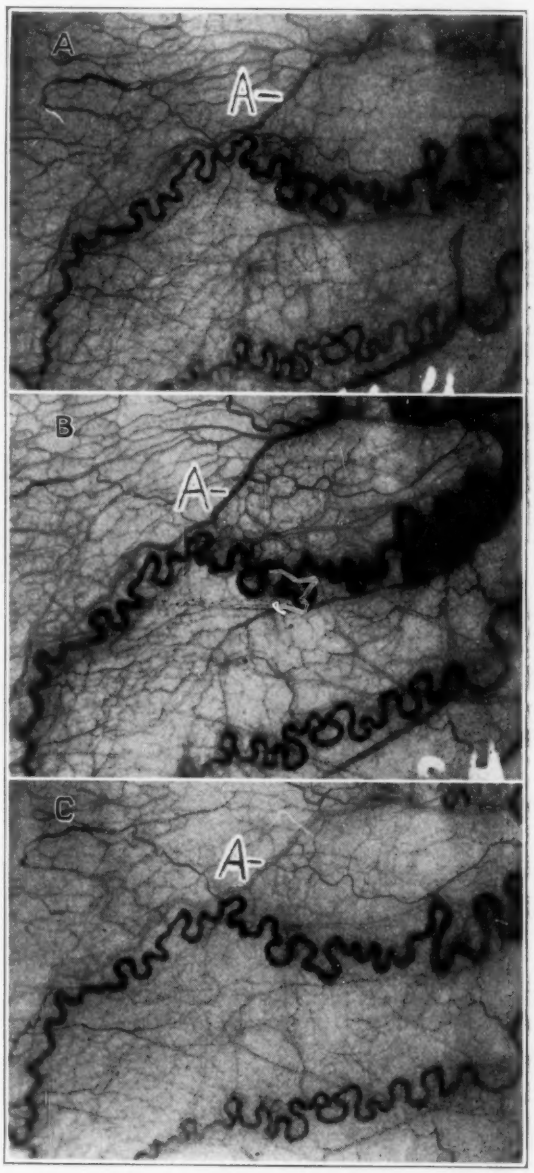

Fig. 9.—Reaction of the conjunctival vessels to pilocarpine in a woman aged 72 with glaucoma.

(*a*) Before medication, with (*A*) an aqueous vein showing layering of the blood and aqueous. (*b*) Two minutes after instillation of a 2 per cent solution of pilocarpine hydrochloride into the conjunctiva. Note the moderate vasodilation of all the blood vessels. The aqueous vein shows pronounced diminution of the aqueous layer. (*c*) Twenty-three minutes after instillation of pilocarpine. The arteries and veins are nearly of the same size as before pilocarpine was instilled, and fewer capillaries are evident. The aqueous vein shows a notable increase in its aqueous content. × 12.6.

19. Peshkin, M. M.: Dry Pollen Ophthalmic Test in Pollen Asthma and Hay Fever Patients Negative to Cutaneous Tests, J. Allergy **3**:20, 1931.

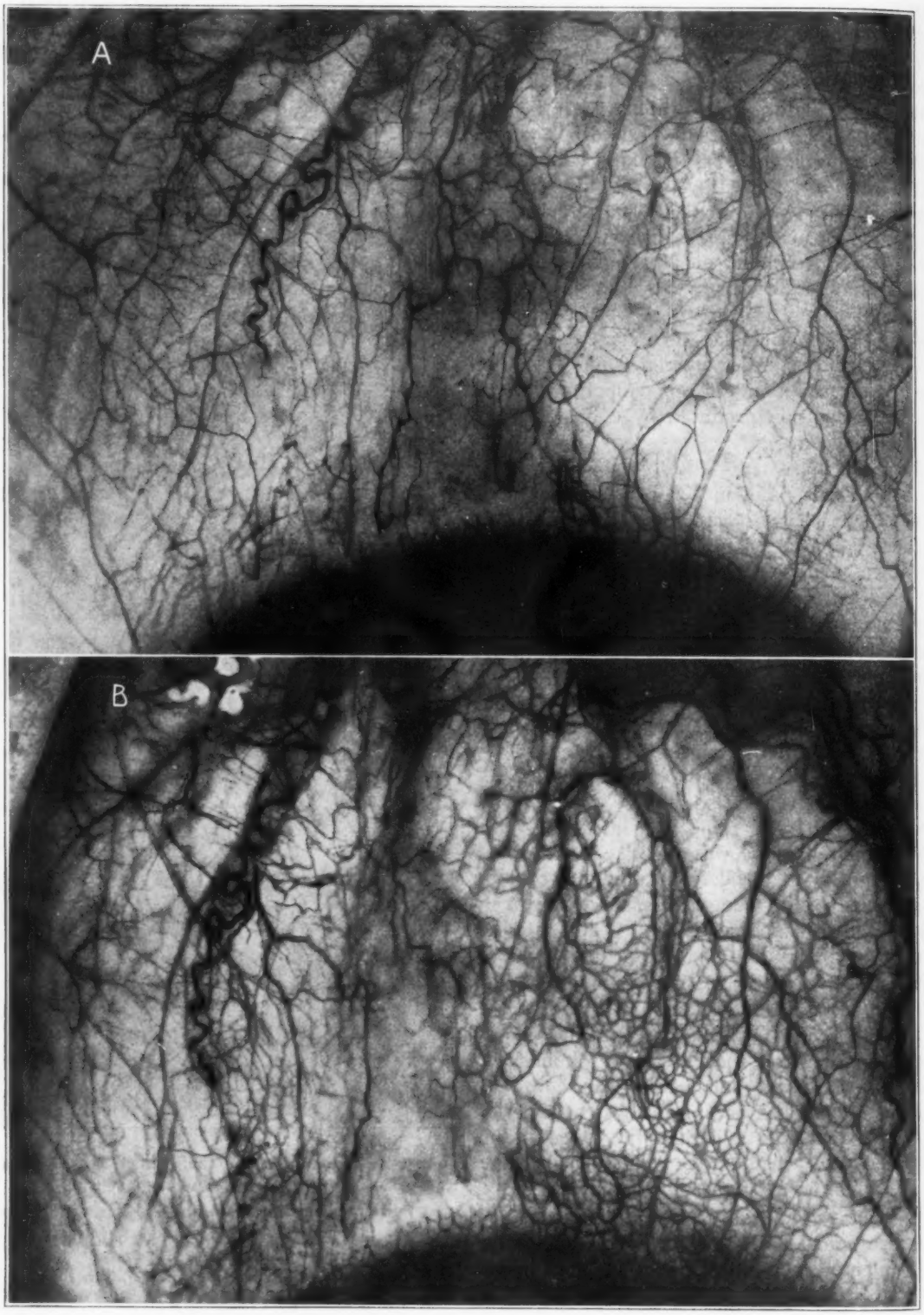

Fig. 10.—Reaction of the conjunctival vessels to histamine in a woman aged 53. *A*, before medication, and *B*, sixty-five seconds after instillation of a solution of biphosphate histamine (1 : 50,000), showing notable vasodilation. × 13.3.

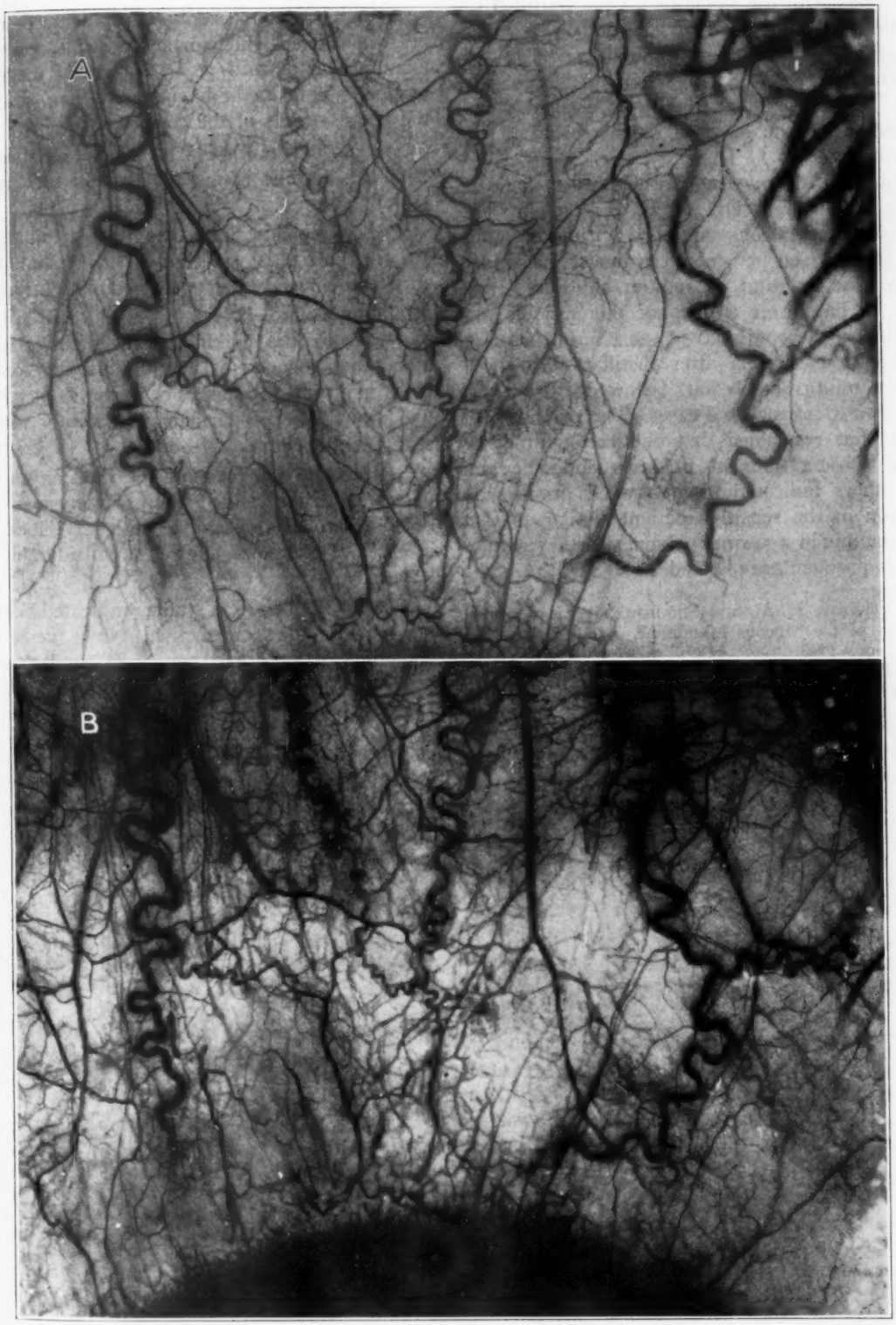

Fig. 11.—Reactions of conjunctival vessels to ragweed pollen in a man aged 44 with pulmonary tuberculosis and hay fever due to ragweed. *A*, before medication, and *B*, forty seconds after instillation of ragweed pollen into the conjunctiva, showing notable vasodilation, similar to the histamine reaction. × 16.

Feldman and Sherman [20] made interesting observations with the slit lamp on the conjunctival reaction to allergens.

Ratner [21] stated that stimulation of smooth muscle is the universal reaction in allergic persons. He presented a theory to explain wheal formation on the basis of initial spasm of the arterioles.

Studies with ragweed antigen were made in collaboration with Dr. D. E. Silberman. The ragweed preparations contained 0.1 and 0.01 mg. of total nitrogen per cubic centimeter suspended in isotonic solution of sodium chloride. In 4 cases of hay fever a drop of this extract was instilled in the conjunctival sac. Serial pictures, starting ten seconds after instillation, demonstrated mild vasodilation, followed by gradual recession to normal in 3 cases. The reaction was milder but resembled that obtained with histamine. Observations of Feldman and Sherman and others indicated that ragweed produces a reaction in the sensitive patient similar to that of histamine in a normal person. Further studies on this problem are planned.

20. Feldman, L. A., and Sherman, H.: Hypersensitiveness of the Mucous Membrane, Arch. Ophth. **29**: 989 (June) 1943.

21. Ratner, B.: Allergy, Anaphylaxis and Immunotherapy, Baltimore, Williams & Wilkins Company, 1943.

SUMMARY

A high speed macrophotographic technic has been developed which makes possible a detailed examination of the eye by means of magnified pictures. This method was used in a study of the blood vessels of the conjunctiva.

Studies of the capillaries of the conjunctiva with this technic opens a new field for investigation.

The diagnostic value of the limbal capillaries in ariboflavinosis is questioned. It should be checked by means of a control test with histamine.

The aqueous veins contain a mixture of blood and aqueous in a striking arrangement of layers. This phenomenon makes possible the location of large drainage channels of the aqueous. These vessels may be of particular importance in the study of glaucoma.

Studies on the reactions of the conjunctival vessels to drugs yielded interesting results. The reactions to physostigmine and to pilocarpine in cases of glaucoma showed a considerable difference.

The results of a few studies indicated that the reaction of the conjunctival blood vessels to histamine in a normal person is similar to the reaction to allergens in an allergic patient.

521 Park Avenue.

BINOCULAR PAPILLEDEMA IN A CASE OF TORULOSIS ASSOCIATED WITH HODGKIN'S DISEASE

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NEW YORK

The absence of any report in the ophthalmologic literature on the disease known as torulosis, especially its association with Hodgkin's disease, has prompted the presentation of this case. Before describing the case, I shall state briefly the main factors concerned in the two diseases and their association.

As early as 1861 a yeastlike organism causing symptoms of disturbance of the central nervous system was mentioned in the literature.[1] In 1916 Stoddard and Cutler[2] differentiated the pathogenic yeast organism from true yeast and named it Torula histolytica. By 1942, 78 cases of torulosis had been reported,[3] in a large percentage of which lesions of the fundus existed.[4] It is noteworthy that in at least 10 per cent of the cases the torulosis was associated with Hodgkin's disease.[5] This is too frequent an occurrence to be a mere coincidence, but so far the relationship of the two diseases has not been established.

Read at a meeting of the New York Academy of Medicine, Section on Ophthalmology, May 15, 1944.

From the Department of Ophthalmology of the New York Post-Graduate Medical School and Hospital (Columbia University).

1. Zenker: Encephalitis mit Pilzentwickelung im Gehirn, Jahresb. d. Gesellsch. f. Natur- u. Heilk. in Dresden, 1861-1862, p. 51.

2. Stoddard, J. L., and Cutler, E. C.: Torula Infection in Man, Monograph 6, Rockefeller Institute for Medical Research, 1916.

3. Warvi, W. N., and Rawson, R. W.: Torula Meningitis, Arch. Int. Med. **69**:90 (Jan.) 1942.

4. (*a*) Torula Meningitis; Lymphoblastoma, Hodgkin's Type, Cabot Case 20241, New England J. Med. **210**:1291 (June 14) 1934. (*b*) McKendree, C. A., and Cornwall, L. H.: Meningoencephalitis Due to Torula, Arch. Neurol. & Psychiat. **16**:167 (Aug.) 1926. (*c*) Fitchett, M. S., and Weidman, F. D.: General Torulosis Associated with Hodgkin's Disease, Arch. Path. **18**:225 (Aug.) 1934. (*d*) Ginsburg, S.: Lymphosarcoma and Hodgkin's Disease, Ann. Int. Med. **8**:14 (July) 1934; **10**:337 (Sept.) 1936. (*e*) von Hansemann: Ueber eine bisher nicht beobachtete Gehirnerkrankung durch Hefen, Verhandl. d. deutsch. path. Gesellsch. **9**:21, 1905.

5. (*a*) Wile, U. J.: Cutaneous Torulosis, Arch. Dermat. & Syph. **31**:58 (Jan.) 1935. (*b*) Footnote 4*a*. (*c*) Warvi and Rawson.[3] (*d*) Fitchett and Weidman.[4c]

Torula, a pathogenic yeastlike organism, is widely distributed throughout nature. It is found only rarely in human beings, and then it usually invades the nervous system. The organism differs from true yeast in that it is generally circular and has a sharply defined nucleus surrounded by a gelatinous capsule, which is enclosed in a thin membrane with a fuzzy exterior. Torulas reproduce only by budding and never form endospores or myceliums, as is the case with normal yeast. Torulas ferment sugar only under certain conditions.

The main clinical symptoms of torulosis are excruciating headache, drowsiness and vomiting, frequently accompanied by papilledema with increased intracranial pressure. The portal of entry of the organism into the body has not yet been discovered. The diagnosis is established by frequent smears and cultural examinations of the spinal fluid.

Hodgkin's disease, a more common condition, is characterized histologically by typical cellular and fibrous involvement of the lymph nodes. It rarely affects the central nervous system because of the absence of lymphoid tissue in the brain. There are, however, a few references to intracranial involvement in the literature.[6] The fundus is rarely affected. The clinical manifestations of Hodgkin's disease consist mainly of enlargement of the cervical nodes, but there may also be loss of weight and secondary anemia with lethargy.

The association of these two diseases has long baffled clinicians. According to Fitchett and Weidman,[4c] "Torula histolytica is one of perhaps several micro-organisms which may evoke the histologic picture of Hodgkin's disease." In a case presented by Wile[5a] in which the diagnosis of Hodgkin's disease had been made yeastlike bodies were observed on microscopic examination of a section of a lymph node which had been removed four and a half years before his report, evidence that there had been an early torular infection. On the other hand,

6. Davison, C., and Michaels, J. J.: Lymphosarcoma with Involvement of Central Nervous System, Arch. Int. Med. **45**:908 (June) 1930. Ginsburg.[4d]

Forbus [7] stated the opinion that the resemblance between the two diseases was superficial and that he had repeatedly failed to recover fungus organisms of any kind in cases of Hodgkin's disease.

The treatment of these two diseases, especially when the conditions are associated, has been unsatisfactory. Some patients have seemed to respond to iodide therapy, while others have improved under treatment with a sulfonamide compound, together with lumbar punctures. The latter method suggested to us the initial line of treatment employed in our case.

REPORT OF A CASE

Mrs. E. L., aged 36, with no children, was referred to me because of deteriorated vision of about two months' duration. She had been a clerk in an insurance company for seventeen years but had been unable to work for the last year and a half because of poor health. Her family and personal histories were irrelevant. She complained of severe headaches, accompanied by drowsiness and intermittent vomiting. Her physician reported that her physical examination showed nothing abnormal except for enlarged cervical lymph glands, present for two years. She had been examined the previous April by Dr. Nathan Rosenthal, who noted that the blood picture was suggestive of early Hodgkin's disease. The hemoglobin concentration was 69 per cent; there was a slight increase in the nonsegmented cells, and the lymphocyte count was 22 per cent. The sternal bone marrow was normal except for slightly diminished cellularity. Dr. Paul Klemperer confirmed the diagnosis of early Hodgkin's disease by biopsy of a cervical node, made the following month (fig. 2). Eight roentgen ray treatments were given, to which the glands responded.

An ophthalmologic examination, made on Jan. 10, 1944, gave the following results: Vision in the right eye was 20/200 and was corrected with a +2.5 D. sphere to 20/40. Vision in the left eye was 18/200 and was corrected with a +3 D. sphere to 20/100. The fields of vision and the external appearance of the eyes were normal. A relative color scotoma and a scintillating scotoma were present. The fundus of the right eye showed elevation of the disk of 7 D., with pronounced edema of the surrounding area (fig. 1). The veins were engorged and tortuous and appeared kinked in spots. The arterioles were normal. Minute hemorrhages and exudates were noted in the disk. A partial star figure was visible in the macular area. The changes in the fundus of the left eye were similar to those in the right eye except that the lesions were more pronounced and the elevation of the disk was 8 D.

Since papilledema as a rule does not accompany Hodgkin's disease, I was prompted to investigate further the etiologic factor in the prominent binocular elevation of the disks, with cerebral sypmtoms. The patient was therefore sent to the New York Post-Graduate Hospital on January 17 for a complete clinical study, in order to exclude the possibility of a cerebral tumor, tuberculous meningitis or syphilis. At this time her temperature, pulse and blood pressure were normal, and the blood culture yielded no organisms.

An electroencephalogram showed an abnormal potential in the right occiput, suggestive of associated increased intracranial pressure. Results of neurologic tests, made by Dr. George Blakeslee, were essentially negative. The papilledema and the results of the electroencephalographic test suggested to Dr. Blakeslee the diagnosis of Hodgkin's disease with lymphatic infiltration into the posterior fossa, resulting in meningoencephalitis.

Lumbar puncture revealed that the cerebrospinal fluid pressure as registered by the water manometer was 600 mm., the average normal pressure being 150 mm. After withdrawal of 15 cc. of spinal fluid, the final pressure was 400 mm. A few hours later the headache was relieved, and the swelling of the disks decreased about 2 D. The spinal fluid was clear; the cell count revealed 65 white cells per cubic millimeter, with 15 per cent polymorphonuclear leukocytes, 77 per cent lymphocytes and 8 per cent unrecognized cells. Unfortunately, no smears or cultures of the spinal fluid were made at this time. The Wassermann reaction was negative.

Dr. William H. Meyer reported that roentgenographic examination of the skull and a detailed survey of the thorax revealed nothing of significance. Because of the intracranial involvement, radiation therapy to the skull was instituted but was discontinued after four treatments as being ineffective.

After two weeks the patient's general condition seemed improved, and she was discharged from the hospital and referred to her family physician.

An ophthalmic examination, on February 14, showed improvement in visual acuity. Vision was 20/40 in the right eye, with correction to 20/20, and 18/200 in the left eye, with correction to 20/50. The swelling of the disks had subsided about 1 D. in each eye. A slight exudate was still apparent in the macula of the left eye; otherwise, the condition was the same as that noted in previous examinations.

A second electroencephalogram, made the following day, showed a decrease in intracranial pressure, in addition to lessening of abnormal transmission over the occiput. As the excruciating headaches and the papilledema persisted, the patient was readmitted to the Post-Graduate Hospital, on February 23. A second lumbar puncture revealed clear spinal fluid, under a pressure of 600 mm. of water. The cell count revealed 298 white cells per cubic millimeter, with 5 per cent polymorphonuclear leukocytes, 90 per cent lymphocytes and 3 per cent unrecognized cells. At this time a culture of the spinal fluid, made on Sabouraud's medium,[8] revealed budding yeast cells which were identified as torulas (fig. 3). The diagnosis was confirmed by many subsequent cultures. This observation was a great surprise and called for a complete reexamination of the patient, since it was now realized that two serious diseases must be considered.

The patient was placed under the care of Dr. Maurice Bruger, who administered sulfadiazine, at first orally and then intravenously, as an inhibiting agent to the torular infection. There was evidence of the sulfadiazine in the spinal fluid to the amount of 7.1 mg. per hundred cubic centimeters, indicating the diffu-

7. Forbus, W. D.: Reaction to Injury, Baltimore, Williams & Wilkins Company, 1943, pp. 744-746.

8. Miss Gladys Morton, of the bacteriologic laboratory, made the culture of the spinal fluid.

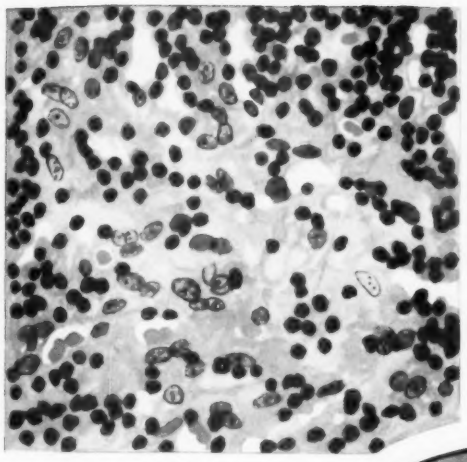

Fig. 2.

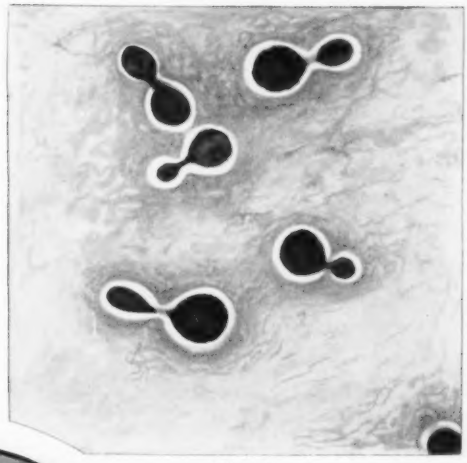

Fig. 3.

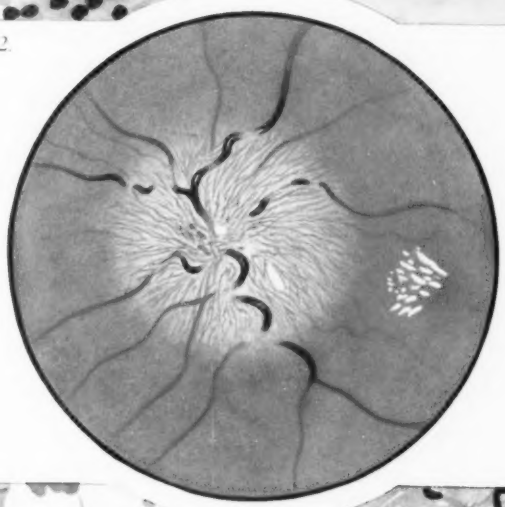

Fig. 1.

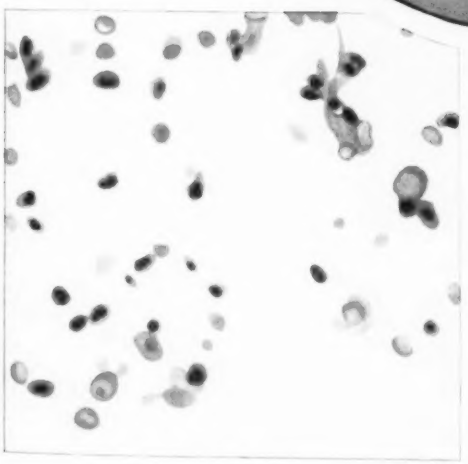

Fig. 4.

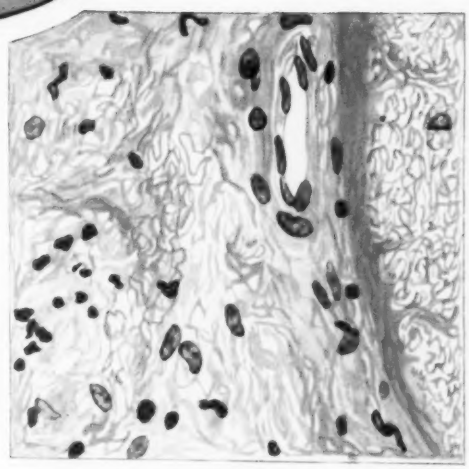

Fig. 5.

1, fundus of the right eye, showing elevation of the disk and edema of the surrounding area; *2*, section of a cervical lymph node with Hodgkin's disease; *3*, torulas cultured from the spinal fluid; *4*, in vitro growth of torulas, showing absence of inhibitory effect of penicillin, and *5*, pericellular infiltration of the pia-arachnoid of the optic nerve.

sion of the drug to the central nervous system. The use of penicillin was considered but was decided against, since in vitro experiments had demonstrated no inhibitory powers of the substance, an observation substantiated by animal experiments performed by Dr. J. Ward MacNeal (fig. 4). As the patient had become emaciated and greatly weakened from constant vomiting, a 5 per cent solution of dextrose with amino acid was injected intravenously.

Since it was feared that the intracranial pressure, associated with papilledema, might produce blindness with secondary optic nerve atrophy, bioccipital decompression was suggested. However, ventricular drainage was considered the safer procedure because of the extremely high pressure of the spinal fluid. The operation, performed with local anesthesia by Dr. Carl Peterson on March 2, consisted of trephination of the skull for drainage of the ventricles. A perforation was made on the right side, about 7 cm. above the external occipital protruberance. The cortex was observed to be under extreme pressure, protruding through the dura. A catheter was inserted into the ventricle, the end being attached to a calibrated sterile bottle by means of rubber tubing. The bottle was elevated 11 cm. above the patient's head to maintain normal pressure. In order to test the advisability of continuing the drainage, a clamp was placed on the connecting rubber tube, preventing the flow of fluid to the bottle. The patient's vision was then greatly reduced; her headaches became more severe, and the papilledema increased. Two hours after the clamp was released vision returned, and the appearance of the fundi was similar to that noted before the operation. This proved the mechanical effect of increased intracranial pressure on the optic disks. At this time it was noted that the pressure in the ventricles equaled that in the spinal canal, an indication of freedom of passage to the latter. Ventricular drainage was continued for a week, about 300 cc. of fluid being recovered daily. Smears and cultures of the ventricular fluid revealed T. histolytica and sulfadiazine to the amount of 5.9 mg. per hundred cubic centimeters. On March 7 cultures of the urine disclosed torulas.

At the end of a week ventricular drainage was discontinued. The spinal fluid was now under a pressure of 240 mm. of water. After 10 cc. was withdrawn the pressure was normal. The patient's vision improved to 20/30 in the right eye and to 20/70 in the left eye; but her headaches persisted, and fresh hemorrhages and exudates were perceptible in the fundi.

An unfortunate complication now occurred. Cultures of the spinal fluid showed a superimposed infection of nonhemolytic streptococci. The temperature rose to 103 F.; the cell count showed increase to 2,075 white cells per cubic millimeter, with about 49 per cent polymorphonuclear leukocytes, and the spinal fluid became cloudy. Henceforth the streptococci, as well as the torulas, persisted in all cultures of the spinal fluid, though to a far less notable degree. Injections of methylrosaniline chloride were now instituted, 1 per cent being given intraspinally because of the fungicidal properties of this drug, but the inhibitory effect on the torulas was not evident.

The patient's vision now showed severe deterioration. On March 29 light perception was zero in both eyes; the pupils were of normal diameter but did not react to light. Examination of the fundi on April 4 showed grayish disks; the margins were completely blurred, with edema of the surrounding area. There was no elevation of the disks, and the retinal veins were less engorged. This improvement can be explained by the normal retrogression of a severe pathologic condition.

The patient's general condition became progressively more serious. The neck became rigid, and a state of opisthotonos gradually developed, with tremors of the upper extremities. On April 18 she passed into a state of coma and died of respiratory and cardiac failure.

Autopsy.—Microscopic examination of the thoracic, abdominal and cervical lymph nodes revealed Hodgkin's disease. No torulas were seen in sections of the lymph nodes.

Torulas were demonstrated in the brain tissue, as well as in the secretions covering the cortex of the cerebrum and the pons, with production of leptomeningitis at the base of the brain. Edema of the brain was present, with dilatation of the ventricles. The aqueduct of Sylvius was partially occluded by an exudate. These pathologic changes were the result of the tortular infection.

Pericellular infiltration of the pia-arachnoid of the optic nerve was demonstrated (fig. 5). This condition may have been an additional factor in production of the binocular papilledema.

SUMMARY

The unusual feature of this case was the pronounced binocular papilledema with cerebral symptoms in association with Hodgkin's disease.

Lumbar puncture revealed that the spinal fluid was under extreme pressure. After subsequent lumbar puncture, cultures of the spinal fluid disclosed the presence of T. histolytica, the causal agent of torulosis.

The discovery of the torulas gave a different aspect to the patient's general condition. The relationship of the two diseases, torulosis and Hodgkin's disease, could not be determined.

The prognosis of either Hodgkin's disease or torulosis is unfavorable; it is even more grave when both the diseases are present.

A complete autopsy confirmed the clinical diagnosis of Hodgkin's disease associated with torulosis.

It is important that smears and cultures of the spinal fluid and of the urine be made in the early stages of all diseases of the brain, especially if cerebral tumor, meningitis and syphilis can be excluded.

29 East Sixty-Fourth Street.

KRUKENBERG SPINDLE AND ITS RELATION TO ANNULAR PIGMENTED BAND ON PERIPHERY OF THE LENS

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A pigmented spindle of the cornea was first described by Krukenberg [1] in 1899; he stated the belief that it arose as a result of fusion between the pupillary membrane and the cornea in fetal life. He referred to the condition as congenital melanosis of the cornea. This abnormality is a vertical band on the posterior surface of the central portion of the cornea composed of brown pigmented particles, which are dense in the center and thin out at the ends and edges. A scattering of fine, dustlike granules is seen outside the spindle, particularly inferiorly. The spindle is usually centrally located; less commonly it is slightly nasal or temporal.

The Krukenberg spindle is a rare anomaly. Evans, Odom and Wenaas [2] were able to collect 202 cases, only a small number of which had been reported. They stated that many busy ophthalmologists have never observed a Krukenberg spindle. Further, many of the atypical varieties that have been reported were probably nothing but the common deposits of pigment frequently seen on the posterior surface of the senile cornea, caused by degenerative and inflammatory changes. Evans and associates found only 7 reported cases of Krukenberg spindle associated with an annular pigmented ring of the lens, 1 of which was described by an American ophthalmologist (Zentmayer [3]). Recently Cameron [4] reported another case. In 78.8 per cent of the reported cases the spindle was present bilaterally, and in 80 per cent it occurred in persons over 30 years of age. This condition shows a distinct predisposition to occur in females (62.3 per cent of patients were female and 37.7 per cent males). In these respects there is a great similarity between Krukenberg spindle and retroiridal pigmented lines on the anterior capsule of the lens; both are rare pigmentary anomalies occurring most frequently in middle-aged females. In young persons under 30 the sex incidence of Krukenberg spindle is reversed, 62 per cent of the patients being males and 38 per cent females. Frequently ocular disease, such as myopia, inflammatory phenomena, pigmentary degeneration of the iris, opacities of the lens, glaucoma and detachment of the retina, is associated with this condition.

The mechanism for the formation of the pigmented band is not clearly understood. The older authors stated that the spindle was a congenital pigmentary abnormality, but the more modern writers have expressed the belief that it is an acquired condition. Goldberg [5] and Moeschler [6] showed that pigment set free by degenerative changes in the iris could be transported to the posterior surface of the cornea by currents in the aqueous. It is likely that alterations in the corneal endothelium must take place before the fine pigment granules can adhere to the cornea (Kraupa,[7] von Sallmann,[8] Gifford,[9] Korobova,[10] Goar [11] and Puglisi-Duranti.[12] Of

1. Krukenberg, F.: Beiderseitige angeborene Melanose der Hornhaut, Klin. Monatsbl. f. Augenh. **37**:254-258, 1899; Weitere Mittheilung über angeborene doppelseitige Melanose der Hornhaut, ibid. **37**:478-480, 1899.

2. Evans, W. H.; Odom, R. E., and Wenaas, E. J.: Krukenberg's Spindle, Arch. Ophth. **26**:1023-1056 (Dec.) 1941.

3. Zentmayer, W.: Association of Annular Band of Pigment on the Posterior Capsule of the Lens with Krukenberg Spindle, Arch. Ophth. **20**:52-57 (July) 1938.

4. Cameron, W.: Krukenberg Spindle Associated with Megalocornea and Posterior Pigmentation of the Lens, Am. J. Ophth. **24**:687-689, 1941.

5. Goldberg, H.: Pigmentkorperchen an der Hornhautinterfläche, Arch. f. Augenh. **58**:324-336, 1907.

6. Moeschler, H.: Untersuchungen über Pigmentierung der Hornhautrückfläche bei 395 am Spaltenlampmikroskop Untersuchten Augen gesunder Personen, Ztschr. f. Augenh. **48**:195-202, 1922.

7. Kraupa, E.: Studien über die Melanosis des Augapfels, Arch. f. Augenh. **82**:67-93, 1917.

8. von Sallmann, L.: Ueber eine dystrophische Endothelveränderung und Pigmentierung der Hornhautrückfläche, Ztschr. f. Augenh. **58**:348-357, 1926.

9. Gifford, S. R.: Epithelial Dystrophy of the Cornea and Its Relation to Endothelial Dystrophy, Am. J. Ophth. **9**:81-85, 1926.

10. Korobova, V.: On the Etiology of Krukenberg's Spindle, Rusk. opht. j., April 1929, pp. 476-484; abstracted, Am. J. Ophth. **12**:699 (Aug.) 1929.

11. Goar, E. L.: Congenital Pigmentation of the Cornea (Krukenberg Type), Tr. Am. Ophth. Soc. **25**: 346-353, 1928; Dystrophy of the Corneal Endothelium (Cornea Guttata) with Report of Histological Examination, Am. J. Ophth. **17**:215-221, 1934.

12. Puglisi-Duranti, G.: Pigmentazione a fuso della faccia posteriore della cornea (fuso de Krukenberg), Arch. di ottal. **42**:183-197, 1935.

further interest is the fact that endothelial dystrophy of the cornea occurs three times as frequently in women as in men (Goar [11]). The greater frequency of the Krukenberg spindle in females, particularly women past the menopause, supports the theory that an endocrine dysfunction plays a role in its formation. The corneal pigment is derived either from degenerative changes in the iris or, if the Krukenberg spindle is associated with a pigmented ring on the periphery of the lens, from the pigmented markings on the surface of the lens.

CASE 1.—A seaman aged 28 was seen at an Army hospital on March 20, 1944 for a routine ocular examination. There were no complaints referable to the eyes. The general physical condition was excellent. Visual acuity was O. D. 20/50 + 2 in the right eye and O. S. 20/20 in the left eye. The irises were blue. Refraction with homatropine cycloplegia revealed an error of + 0.75 D. sph. ⌒ + 0.25 D. cyl., axis 45 in the right eye and of + 0.25 D. sph. ⌒ + 0.75 D. cyl., axis 180 in the left eye. Visual acuity with correction was 20/40 in the right eye and 20/20 in the left eye. With oblique illumination a fusiform streak was observed on the posterior surface of the cornea. It was slightly nasal to the pupillary center and measured 6 mm. in length and 1 mm. at its greatest width. It extended downward from a point opposite the upper pupillary margin. On examination with the slit lamp, the streak resolved itself into numerous fine brown

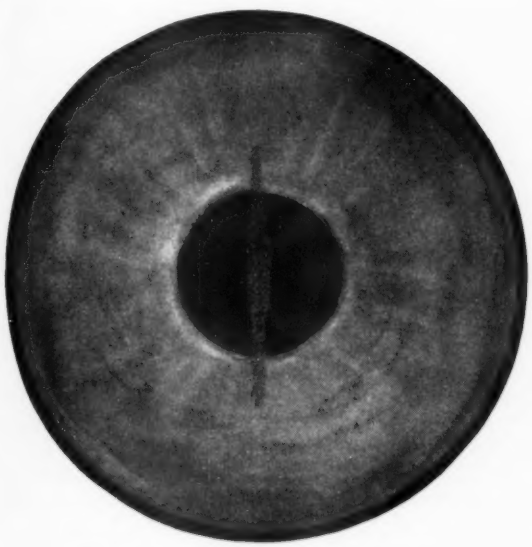

Fig. 1 (case 1).—Krukenberg spindle.

particles, the latter being more numerous in the center and fading away at each end. The particles extended over a greater area than was suspected on examination with oblique illumination alone: Inferiorly the pigmented granules were seen to extend downward as far as visibility permitted; nasally a scattering of pigment was observed for a distance of 1 mm. Careful examination of the anterior chamber revealed two brown pigmented floating particles. Similar particles were present on the anterior surface of the lens and near the pupillary border of the iris. The remainder of the eyeball appeared normal. The left eye showed changes in the iris similar to those in the right eye, but a pigmented spindle was absent. The moderate decrease in visual acuity could not be explained by the objective findings.

On April 4 the patient returned for a postcycloplegic test and determination of the visual fields. At this

Fig. 2 (case 2).—Krukenberg spindle, with a pigmented, annular ring in the periphery of the lens.

time the posterior surface of the left cornea presented a Krukenberg spindle 3 mm. long and about 0.75 mm. wide. It also was slightly nasal to the pupillary center. Inferior to the spindle a fine scattering of pigment was observable as far down as the limbus. The corneal pigmentation of the right eye had changed from the form of a spindle to that of a club, with the large bulge directed inferiorly.

CASE 2.—A nurse aged 21, who was undergoing physical examination for service in the Army, was first seen by me on April 4, 1944. The family history was without significance. The general physical condition was normal. The irises were blue. Visual acuity was 15/200 in the right eye and 16/200 in the left eye. With a correction of — 2.50 D. sph. ⌒ + 0.50 D. cyl., axis 90 vision was 20/25 in the right eye, and with a correction of — 2.75 D. sph., ⌒ + 0.50 D. cyl., axis 90, it was 20/30 in the left eye. Oblique illumination revealed a Krukenberg spindle bilaterally, each lying slightly nasal to the pupillary center and measuring 3 mm. in length and 1 mm. in width. Observation of each eye with the slit lamp disclosed that the greatest collection of pigment was in the center, the concentration decreasing at each end. Superiorly pigment was not observed, but inferiorly a scattering of pigment extended from the tip of the spindle to the limits of visibility. Similar pigment granules were present on both surfaces of the lens and on the anterior surface of the iris. In the anterior chamber and the retrolental space a few pigmented particles were also present.

After complete dilation of the pupil, a dense brown pigmented ring was visible around the entire circumference of the lens, almost coinciding with the margin

of the completely dilated pupil. In each crystalline lens numerous white, punctate opacities, forming a zonular cataract, were present under the adult nuclear band. Near the posterior pole a yellowish white plaque was present immediately under the capsule.

COMMENT

The unusual features of the 2 cases presented in this report were the discovery of a rare pigmentary abnormality of the cornea (Krukenberg spindle) in 2 young persons in an Army hospital within less than one week; the formation of a spindle in the second eye within six days in case 1 and the rare association of the Krukenberg spindle with a pigmented ring about the periphery of the lens in case 2. The presence of fine pigment granules on the surface of the iris and similar particles on the anterior surface of the lens and in the aqueous humor (case 1) supports the theory that the pigmentation of the cornea was secondary to the disintegration of the iris pigment. The unusual pigmentation of the periphery of the lens in the second case was the probable source of the pigment seen on the cornea, the surfaces of the lens, the retrolental space and the aqueous humor. Mann [13] stated the belief that the pigmented markings at or near the periphery of the lens are due to an abnormal maintenance of the contact between the tips of the ciliary processes and the lens in early life. It is usually stated that the particles reach the corneal surface through transportation by the thermal currents in the aqueous. Another possibility, however, is that the migration may be the result of electrical currents in the eye (Bellows [14]).

SUMMARY AND CONCLUSIONS

Two cases of Krukenberg spindle were observed in an Army hospital in a period of six days. In 1 case the condition was observed to be unilateral at the first visit and bilateral at the second examination, six days later. In the second case the pigmented spindle was associated with an annular pigmented band on the periphery of the lens.

13. Mann, I.: Developmental Abnormalities of the Eye, London, Cambridge University Press, 1937.

14. Bellows, J.: Cataract and Anomalies of the Crystalline Lens, St. Louis, C. V. Mosby Company, 1944.

PIGMENTED LINES IN RETROIRIDAL REGION OF ANTERIOR CAPSULE OF LENS

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Pigmented lines on the lens were first described by Brueckner [1] in 1907. Vogt [2] referred to this condition as retroiridal pigmented lines on the anterior capsule of the lens and described 9 cases in his "Atlas." Lugli [3] and Streiff [4] reported 5 and 3 cases respectively. Twenty-two cases in Geneva, Switzerland, were reported by Bischler.[5] As far as I can determine, this abnormality has never been described in the American or the English literature. The purpose of this report is to bring the condition to the attention of American ophthalmologists and to report 2 new cases, 1 of a soldier aged 27. His case is unique because in all previously reported cases the condition occurred in persons over 40 years of age.

Pigmented lines in the retroiridal region of the anterior capsule of the lens occur nearly always bilaterally, although they may be more pronounced on one side than on the other. In Bischler's series the condition was bilateral in 19 of 22 cases. In table 1 are stated the ages at which this abnormality has been observed.

TABLE 1.—*Pigmented Striae on the Capsule of the Lens in Relation to Age*

Author	Year of Report	Age, Years	
		Range	Average
Vogt [2]	1921 and 1931	40-73	55
Lugli [3]	1933	53-77	64
Streiff [4]	1935	62-63	..
Bischler [5]	1939	44-74	60
Bellows	1944	27-57	..

The pigmented lines are seen far more frequently in females than in males (table 2). A similar predisposition in females is observed with Krukenberg's spindle. Thus, with respect to age and sex there is a notable similarity between the two pigmentary abnormalities (Bischler,[5] Evans and associates [6] and Bellows [7]).

TABLE 2.—*Pigmented Striae on the Capsule of the Lens in Relation to Sex*

Author	No. of Cases	Females	Males
Vogt	9	7	2
Lugli	4*	1	3
Streiff	3	3	0
Bischler	22	18	4
Bellows	2	0	2

* In 1 case the sex was not specified.

Vogt asserted that the radially arranged pigmented lines on the anterior lenticular capsule are vestigia of the capsulopupillary membrane. He offered as proof of this hypothesis 3 cases in which he noted other pigmented remnants of the pupillary membrane. He pointed to the fact that pupillary remnants derived from the ciliary arteries are pigmented, while remnants of the tunica vasculosa in the posterior region, which are derived from the arteria centralis, are colorless. Lugli supported Vogt's claims with regard to the origin of the pigmented lines. Streiff expressed the belief that a congenital formation of unpigmented lines exists in cases of this abnormality, favoring the deposition of pigment released by the mechanical friction of the iris on the anterior capsule of the lens. In agreement with Streiff, Bischler, who made observations in some cases over a period of several years, noted changes in the number and size of the pigmented striae. For example, in 1 case the pigmented lines disappeared in one and

1. Brueckner, A.: Ueber Persistenz von Resten der Tunica vasculosa lentis, Arch. f. Augenh. (supp.) **56**: 5-149, 1907.

2. Vogt, A.: Atlas der Spaltlampenmikroskopie des lebenden Auges, Berlin, Julius Springer, 1921; Lehrbuch und Atlas der Spaltlampenmikroskopie des lebenden Auges, ed. 2, ibid. 1930.

3. Lugli, L.: Sui resti della "tunica vasculosa lentis retroiridalis" all' osservazione biomicroscopica, Rassegna ital. d'ottal. **2**:1334-1347, 1933.

4. Streiff, E. B.: Indagine biomicroscopica sui residui delle tunica vascolare retroiridea del cristallino (linee pigmentate retroiridee di Vogt), Atti d. Cong. d. Soc. oftal. ital., 1935, pp. 89-94.

5. Bischler, V.: Ueber die Natur der Vogtschen Streifen (retroiridale Pigmentlinien der Vorderkapsel) an Hand von 22 neuen Fällen, Klin. Monatsbl. f. Augenh. **103**:517-524, 1939.

6. Evans, W. H.; Odom, R. E., and Wenaas, E. J.: Krukenberg's Spindle, Arch. Ophth. **26**:1023-1056 (Dec.) 1941.

7. Bellows, J. G.: Krukenberg Spindle and Its Relation to Annular Pigmented Band on the Periphery of the Lens, Arch. Ophth., this issue, p. 480.

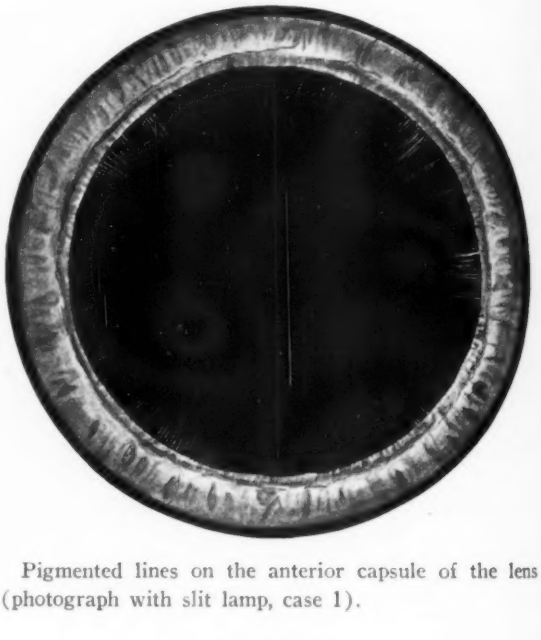
one-half years and in another in four years. He maintained that such changes could hardly be expected in a congenital condition which has persisted into old age and he concluded that the pigmentation is acquired, not congenital. Further support of his contention is the frequency of disintegration of the iris pigment in elderly persons, an occurrence which would readily explain the source of the pigment seen on the capsule of the lens.

The radial pigmented lines can be seen only with the slit lamp and usually require mydriasis. The lines are delicate and radially directed, and if present in all sectors they form a wreath. The size of the striae is variable. They lie midway between the equator and the anterior pole of the lens, usually in the retroiridal region. The total number of lines ranges from less than ten to several hundred; in some sections they lie close together, and in others only a solitary line may be present. There is a tendency for the lines to be more numerous nasally than temporally. Associated ocular signs in this condition are unimportant, although atrophy of the pigmented portion of the pupillary margin of the iris is common. Of less frequent occurrence are persistent pupillary remnants, senile exfoliation of the capsule of the lens and Krukenberg's spindle. To be differentiated from retroiridal pigmented lines are the radially arranged unpigmented lines which Schläpfer and Vogt stated were characteristic of glassblower's cataract, but which Abramowicz,[8] who noted them in 90 per cent of 240 persons who had never been exposed to heat, maintained were merely the result of continuous contact of the rough posterior surface of the iris with the capsule of the lens.

REPORT OF CASES

CASE 1.—A gardener aged 57 consulted me because of difficulty in reading with his old glasses. Visual acuity was 20/50 in the right eye and 20/70 in the left eye. With a correction of + 3.50 D. sph. ⊃ + 1.00 D. cyl., axis 180 for the right eye vision was 20/20 in that eye and with a correction of + 3.25 D. sph. ⊃ + 1.50 cyl., axis 180 for the left eye vision was 20/25 in that eye. With the addition of a + 2.50 D. sphere the patient read Jaeger test type 1 with each eye. After dilation with eucatropine, examination was made with a slit lamp. On the anterior capsular surface of each lens were delicate brown radial streaks, forming a wreath. All the pigmented lines were peripheral to the margin of the undilated pupil. A few punctate opacities were observed in the cortex of the lens. Otherwise each eyeball was normal. The intraocular tension was normal.

CASE 2.—A soldier aged 27 was seen for routine examination of the eyes. Visual acuity was 16/200 in the right eye and 12/200 in the left eye. With a correction of —2.75 D. sph. ⊃ + 0.75 D. cyl., axis 90 for the right eye vision was 20/20 in that eye and with a correction of — 2.50 D. sph. ⊃ + 0.75 D. cyl., axis 90 for the left eye vision was 20/20 in that eye. In the retroiridal region of the anterior capsule of the lens of the right eye delicate lines, some of which were slightly pigmented and others grayish, formed a complete wreath. The anterior capsule of the left eye showed similar, but less distinct, lines. Except for fine punctate opacities in the lens under the band of disjunction, the eyes were normal.



Pigmented lines on the anterior capsule of the lens (photograph with slit lamp, case 1).

COMMENT

In 2 cases of pigmented lines in the retroiridal region of the anterior capsule of the lens reported here the patients were men, and the eyeballs showed no other abnormality. One patient was 27 years of age, the youngest person with this condition whose case has been reported up to this time. The observations tend to support Vogt's hypothesis that the pigmented lines are a vestigium of the capsulopupillary membrane. Yet it is quite possible, as Streiff and Bischler contended, that nonpigmented lines, which are remnants of the pupillary membrane, may become pigmented in elderly persons as a result of disintegration and freeing of the pigment of the iris.

8. Abramowicz, I.: Opacifications striées coronaires superficielles du cristallin, Ann. d'ocul. **170**:602-604, 1933.

DACRYOCYSTITIS

THE TRANSPLANTATION OPERATION

HAROLD GIFFORD JR.
OMAHA

The problem of dacryocystitis is not one for the ophthalmologist or the rhinologist alone, but for both specialists. The patient's first complaint is that of tearing. This usually brings him to the ophthalmologist. There has been too much dissension over who is the proper authority. It is my opinion that cases of this condition are primarily the ophthalmologist's problem, but certainly a rhinologist who has studied the subject is just as capable of handling them. The region is a sort of no-man's land and has been neglected by both groups of specialists. Since the patient's complaint and the complications (conjunctivitis and corneal ulcer) are concerned with the eye, the problem should primarily be that of the ophthalmologist, but he should consult with the rhinologist in every case and with the internist in special instances.

I shall present my concept of the cause and treatment of dacryocystitis, my conclusions having been based on a review of 88 surgical cases of the condition in adults and 37 cases in infants. It is easier to discuss the infantile type first, since this demonstrates the development of the pathologic process concerned with the formation of a typical mucocele in the adult.

As all know, the etiologic factor in the infantile type is the failure of the lower end of the nasolacrimal duct to open, soon after birth. If this end fails to open when lacrimation starts, the sac cannot empty and soon becomes infected. The mucous glands hypertrophy, and a mucocele develops within a month or two. An acute infection rarely complicates this condition. Development of a corneal ulcer in children is also rare. Chronic conjunctivitis, however, is usually present to some degree.

In my series of 37 cases of the infantile type, three interesting trends with regard to treatment are evident (table 1). In 19 cases, or about one-half, cure was effected without surgical intervention. In the other 18 cases the duct was probed, and in 4 of these cases the condition was not cured. In all cases in which cure was not effected probing was done before the age of 6 months. I believe that the lacrimal duct should never be probed before this age. When the lower end of the duct becomes patent, spontaneous cure will take place. I am sure that direct pressure on a full sac exerted down toward the canal will hasten this result. I discovered this procedure myself, but it is probably not new to many here. It is like "the laying on of hands," of medieval days. One, two or three treatments, at weekly intervals, usually results in cure. If this maneuver is not effective, the mother should be advised to empty the sac daily by pressure, and a mild antiseptic (zinc sulfate, ½ grain [0.037 Gm.] to the ounce [31 Gm.]) is prescribed. If tearing and mucus are still present at the age of 6 months, I probe the duct with the child under light ether anesthesia, using only the smallest, no. 1, probe, so that little damage is done to the lining of the lacrimal duct.

TABLE 1.—*Treatment Used in Thirty-Seven Cases of Congenital Stenosis*

	No. of Cases	Cures	No Cure
Probing	18	14	4
Downward pressure	8	8	0
Pressure advised or cure spontaneous	11	11	0

The etiologic factors in the adult form are not quite so simple, but I am sure that the primary cause is obstruction to the flow of tears, either at the nasal end or at the narrowest part of the nasolacrimal duct, just at its upper end, below the sac. Table 2 presents the etiologic factors as well as they could be determined from 125 case histories. The data indicate that a nasal pathologic process was a definite causative factor in a few cases (5 or 6) but that in many cases of the acute form and in some cases of the chronic form infection from the nose was probably an indirect cause. This assumption has never been proved, and since several months are required for completion of obstruction of the duct and for production of a mucocele, acute nasal disease can play only a small part. The

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role of nasal infection is a problem for the rhinologist.

Treatment of the acute form should be much more effective when the sulfonamide compounds and penicillin are used; but if a permanent stricture has been produced, these substances will not prove effective, and the sac will have to be drained surgically. I try to empty the sac by gentle pressure and lavage but do not attempt to probe beyond the canaliculus. The end opening into the sac becomes blocked by kinking and edema and can sometimes be opened by gentle probing. If the sac can be emptied from above without incision, the duct often drains after the infection subsides. If this measure fails, the sac should be incised deeply and sulfanilamide powder dusted in or carried in with a drain. From two to four weeks after healing is complete, the status should be surveyed, and if a mucocele or fistula is present, some type of dacryocystorhinostomy should be done. If the sac has been completely destroyed, nothing further is needed unless tearing is excessive.

TABLE 2.—*Etiologic Factors in One Hundred and Twenty-Five Cases of Dacryocystitis*

	No. of Cases
Anatomic	
Congenital stenosis	37 *
Trauma	
To nasal and facial bones	7
Probing in childhood	8
Operation on nose	1
Nasal pathologic process	
Chronic sinusitis	2
Deviated septum	2
Polyps	1
Infection of sac	
Congenital syphilis	0
Acute infection	14
Chronic infection	24
Syphilis	0
Tuberculosis	1
Tumors	
Lymphatic leukemia	2
Squamous cell epithelioma	1
Insufficient data	23

* The ratio of males to females was 1 to 3. Negroes rarely have obstruction.

An atrophic sac presents the problem of tearing only, since it rarely becomes reinfected and there is no danger to the cornea. This condition should be looked for carefully, inasmuch as my associates and I have operated on several such sacs in an attempt to cure the epiphora, without results. The condition is not included in the usual discussions on dacryocystitis. The diagnosis rests on the previous history of an acute attack of dacryocystitis, or sometimes only on the presence of tearing for a long period without infection. There is definite evidence of obstruction. Saline solution cannot be injected into the nose from either the upper or the lower punctum, and the probe does not enter the sac through the upper or the lower canaliculus. The most important sign is the absence of mucus when the sac is pressed on. In cases of atrophic sac, the Arruga technic may be applied, or the secretory duct from the lacrimal gland may be excised safely if tearing is annoying.

Mucocele offers no real problem. Dacryocystorhinostomy will effect a cure in nearly 90 per cent of cases. The only reason for failure in cases of this type is faulty surgical technic or anatomic anomalies. The sac should not be removed except in cases of tuberculosis, tumor or leukemia. The diagnosis of such a condition should be made at the time or before operation is attempted, and the planned dacryocystorhinostomy can easily be turned into an extirpation.

The basis for the opinions I express here is experience in a series of 85 cases of obstruction of the lacrimal duct in which Dr. W. H. Stokes and I have performed operation in the last nine years (table 3). The technic of the transplantation operation is simple, really much less difficult than is removal of the sac. We use a plan similar to that which Burch [1] described in 1920. The technic may have been original with him, but the idea was first put to test by Speciale-Cirincione,[2] in Italy, in 1904. Toti's [3] work

TABLE 3.—*Results of Transplantation of Tear Sac (Eighty-Five Operations)*

	No. of Cases
Successful operation (no mucus; no tearing)	68
Partial failure (still tearing but no pus)	6
Complete failure (still tearing and mucus)	11

TABLE 4.—*Cause of Failure of Transplantation of Tear Sac*

	No. of Cases
Poor technic	
(*a*) Opening into nose made too far posterior	3
(*b*) Opening into nose made too high, so a dependent pocket was left	2
(*c*) Poor dissection—sac too short to go well into nose	1
Nasal pathologic process	
High deviated septum, not repaired before operation	1
Operation done too soon after acute dacryocystitis, which was a contributing factor	2
Outcome unknown; data insufficient	4

1. Burch, F. E.: Conservation of the Lacrimal Sac: A Method, Tr. Am. Acad. Ophth., 1920, p. 137.

2. Speciale-Cirincione, F.: Sulla dacriorinostomia (secondo Toti), Clin. ocul. **14**:1639, 1913.

3. Toti, M. A.: Nuovo metodo conservatore di cura radicale delle suppurazioni cronische del sacco lacrimale (dacriocistorhinostomia), Clin. mod. **10**:385, 1904.

came out in the same year, and further investigation on the transplantation method was dropped in favor of his method. Forsmark,[4] in Sweden, and Neumayer,[5] in Germany, did the transplantation operation in 1911. After 1920 this procedure was reported on from many countries. Stokes got his inspiration from the work of Stock,[6] published in 1934. I helped Stokes with some of his early operations and realized how much easier this method was than the Toti-Mosher operation, which I had been taught to do in Boston.

The lower end of the sac is freed through an external approach; a trephine opening, 8 mm. in diameter, is made into the nose, and the open lower end of the sac is held in place in the nose by sutures pulled out on the cheek. Dr. Stokes[7] has described the operation in detail in his report on 39 cases in which the method was used. Our recent series includes these cases, with further follow-up data (table 4).

1620 Medical Arts Building.

4. Forsmark, E.: On Dacryocystorhinostomy, Hygiea **73**:1432, 1911.

5. Neumayer, in discussion on von Eicken, C.: Ein neues Verfahren zur Beseitigung von Stenosen des Tränennasenkanals, Verhandl. d. Ver. deutsch. Laryng., 1911, p. 600.

6. Stock, W.: Ueber die Erfolge der Operation der Tränensackeiterung nach Einpflanzung des unteren Endes des Tränensacks in die Nase, Klin. Monatsbl. f. Augenh. **92**:433, 1934.

7. Stokes, W. H.: Transplantation (Implantation) of the Lacrimal Sac in Chronic Dacryocystitis, Tr. Am. Acad. Ophth., 1938, p. 342; Arch. Ophth. **22**:193 (Aug.) 1939.

CHRONIC DACRYOCYSTITIS

TREATMENT FROM THE RHINOLOGIST'S POINT OF VIEW

LAVERNE B. SPAKE, M.D.
KANSAS CITY, KAN.

Treatment of chronic dacryocystitis from the rhinologist's point of view involves classification of the condition into certain types: (1) infection of the sac with intranasal abnormalities; (2) infection of the sac with involvement of accessory nasal sinuses; (3) infection of the sac without intranasal involvement, and (4) recurrent infection of the sac with or without a fistulous tract or stenosis of the duct. The etiologic factor in these cases is generally a suppurative lesion in the anterior ethmoid cells, and recovery will not occur until the cells are removed surgically, even though total extirpation of the sac has been performed.

The first symptom of dacryocystitis is tearing (epiphora). Later there is suppuration (due to bacteria), which is recognized by expression of pus into the conjunctiva on pressure over the lacrimal sac. The sac may dilate and rupture, or it may be incised, with a remaining fistula. The patency of the lacrimal duct should be tested for localization of the stenosis. The use of the sound is indicated in the differential diagnosis. A fistula near the inner canthus is of diagnostic importance. This can be proved by the injection of fluid into the canaliculus. Mucocele of the ethmoid cells or the frontal sinus may cause stenosis of the sac from pressure with tearing, but there will be no purulent discharge from the sac.

ETIOLOGIC FACTORS

The exact pathologic process involved in obstruction of the lacrimal sac is still obscure. According to Kuhnt, in 93.7 per cent of cases the condition is due to nasal disease, and Diggle [1] reported the results of an investigation of the nasal condition of 120 patients with obstruction of the lacrimal sac, as follows:

Nasal Lesion or Abnormality	Incidence, Percentage
Injury	4.1
Breadth of nose and nasal passage	
Narrow	10.8
Wide	13.3
Enlargement of middle turbinate bone (moderate only)	10.0
Misplaced middle turbinate	3.3
Deviated nasal septum	8.3
Enlarged inferior turbinate	1.6
Rhinitis—	
Atrophic	5.8
Hypertrophic	5.0
Simple	1.6
Ethmoiditis	
Closed suppurative	1.6
Cystic	3.3
Nonsuppurative with single polypus	1.5

An injury in the region of the inner canthus, producing fracture of the infraorbital margin or of the frontal process of the maxilla, may cause a mechanical obstruction.

ANATOMIC RELATIONS OF LACRIMAL BONE

The surgical anatomy of the lacrimal bone may be summarized as follows: It articulates with the frontal, ethmoid and inferior turbinate bones and with the maxilla. Intranasally, one must consider its relation to the uncinate process, the hiatus semilunaris, the ethmoid bulla and the agger nasi cells, all of which are anterior to the infundibulum ethmoidale. The two uncinate processes of the ethmoid bone articulate with the lacrimal process of the lacrimal bone and with the corresponding processes of the inferior turbinate bone, which close the bony and the mucous membranous part of the maxillary fontanel. From the point of view of the orbital fossa the lacrimal fossa is either deep or shallow. The lacrimal fossa in its upper half is related to the anterior ethmoid cells; in its lower half, to the middle nasal meatus, and on its orbital surface to the lamina papyracea, the orbital plate of the ethmoid labyrinth. The anterior ethmoid artery enters the ethmoid labyrinth from the orbit about 2 cm. directly posterior to the inner orbital rim, through the anterior ethmoid foramen, which admits the artery and nerve. The lacrimal groove contains the lacrimal sac. The descending process contains the nasal duct and terminates in the inferior nasal meatus, under the inferior turbinate.

The membranous and bony lacrimal nasal duct may be divided into three portions: the superior contracted

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1. Diggle, F. H.: Intranasal Dacryocystostomy for Relief of Lachrymal Obstruction, Brit. M. J. **1**:391 (March 7) 1931.

portion, wholly membranous, and including about 12 mm. of the total length; a second inter-osseous portion, which is the widest portion, and included 15 mm. to 16 mm. of the total length; and a third meatal portion, which is buried in the mucous membrane of the lateral wall of the inferior meatus of the nose, taking up 4 mm. to 6 mm. of the length, and depending upon a multitude of normal variations in its inferior ostium.[2]

TOTI-MOSHER OPERATION

Berlin was the first to remove the tear sac, in 1868. In 1904 Toti presented his operation for destruction of the sac, with a window resection through the lacrimal fossa. In 1921 Mosher published a modification of Toti's technic. Many men have presented a technic of their own; for example, West, Polyak, Toti, Dupuy, White, Clark, Campbell, Weynix, Aubart and Bremona employed the intranasal operation and Halle, Koyler, Zarzyeki and Mosher the external approach. While West's operation is purely endonasal, he has reported recoveries in 90 per cent of his cases.

Patients with chronic suppuration, or with the various clinical conditions caused by stenosis, may or may not be proper subjects for operation. Persistent fistula should always be operated on, no matter how long it may have existed. Whenever an obstructive process has developed and resists all forms of treatment, such as irrigation of the sac and probing of the duct, surgical treatment is indicated. Mucocele of the ethmoid and frontal sinuses must be ruled out by roentgenographic study. Injection of iodized poppyseed oil into the sac aids in the differential diagnosis. The lacrimal sac may be dilated as a result of secondary abscess around the sac. A malignant growth is not uncommon. Tuberculosis and osteomyelitis may be present. The bacteriologic aspect must not be overlooked. Fungous infection is not common.

I wish to present the Mosher-Toti operation, a procedure which is fairly simple and is done under direct vision.

Dr. Mosher,[3] in an address as the guest of honor before the American Academy of Ophthalmology and Otolaryngology in 1937, stated that the operation properly done is successful in 90 per cent of cases. A few ophthalmologists were then doing it. A number of these progressive men, however, were not paying attention to infection of the anterior ethmoid cells or to a high deviation of the septum. Neglect of these two points, he said, is sure to vitiate results of the operation.

2. Spaeth, E. B.: Principles and Practice of Ophthalmic Surgery, ed. 3, Philadelphia, Lea & Febiger, 1944.

3. Mosher, H. P.: Unsolved Problems in Otolaryngology, Tr. Am. Acad. Ophth. **42**:27, 1937.

Ballenger and Ballenger [4] described the operation as follows:

Toti-Mosher Operation.—Mosher has modified Toti's technic by using a straight incision 10 mm. from the inner canthus. The first opening is made through the lacrimal bone rather than through the superior maxilla. Toti saves the mucous membrane before he makes the opening. Mosher resects it with bone. Toti does not remove the wall of the inner duct. Mosher removes it down to the upper rim of the inferior turbinate. Toti occasionally removes the tip of the middle turbinate; Mosher does it in all cases.

Technic.—A general or a local anesthesia may be used. If a local anesthesia is elected the novocain [procaine] is injected along the line of the incision, above the inner canthus and down the inner wall of the orbit. In addition the nose may be packed with 20 per cent cocaine and epinephrine solution.

The lid, eyebrow and upper part of the nose are sterilized. The lids are held together with strips of sterile adhesive.

Any obstructing lesions of the septum should be resected and the anterior tip of the middle turbinate removed before the operation on the sac.

An incision (Mosher) is made about 10 mm. from the inner canthus of the eye, starting at the level of the cartilage of the upper lid and extending down parallel with the posterior edge of the ascending process of the superior maxilla to within 2 or 3 mm. below the lower rim of the orbit. The knife is carried through the soft tissues and the periosteum to the bone.

The periosteum is elevated from the inner wall of the orbit by means of ½ inch [1.27 cm.] flat chisel or a periosteal elevator. The periosteum is elevated to the rim of the orbit, passing into the floor of the lacrimal fossa, thus lifting the sac out of its bed.

The middle fossa of the nose is broken through and the opening enlarged by means of a small-sized biting forceps. The opening should be large enough to admit the little finger.

The nasal wall of the sac is removed by grasping the nasal wall with forceps or a small hemostat, excising by means of scissors or a knife. The orbital wall of the sac is left in place.

The inner half of the wall of the sac is then removed with forceps and scissors. The outer half of the sac is left to protect the common opening of the canaliculi.

The inner wall of the nasal duct is removed by means of a small punch. The mucous membrane of the nose around the bone opening is trimmed, the soft tissues replaced and the skin sutured with dermal sutures.

Nasal packing and drains are unnecessary. The skin sutures are removed in forty-eight hours.

The after-treatment consists in instilling from 8 to 10 drops of liquid petrolatum in the nose night and morning for from ten days to two weeks.

A contraindication to the Toti-Mosher operation is stenosis of the common duct of the canaliculi at its entrance into the sac. The patency may be determined by passing a lacrimal probe or by the use of a radiopaque oil and a roentgenogram.

Spaeth [2] recommended cystectomy in all cases of tuberculosis of the sac and of tuberculous osteomyelitis, malignant growths, chronic atrophic rhinitis and chronic suppurative ethmoiditis.

4. Ballenger, W. L., and Ballenger, H. C.: Diseases of the Nose, Throat and Ear, ed. 8, Philadelphia, Lea & Febiger, 1943.

The last condition should be corrected before cystorhinostomy.

In cases in which suppuration is present, with or without mucocele, or in cases of chronic obstruction without suppuration the Mosher operation is indicated.

I have had successful results in 80 per cent of my series of cases of chronic suppurative dacryocystitis in which the Mosher technic was employed. I believe that cases of this condition are neither strictly rhinologic nor strictly ophthalmologic. Consideration should be given to the most suitable type of operation. Careful attention to the intranasal condition should precede the external surgical procedure. When a cystectomy is indicated, an ophthalmologist should be chosen. The Mosher-Toti operation is in the field of rhinology.

The end results in my experience and in the cases reported in the literature have substantiated the claims of Mosher that his method of attack in properly selected cases is a good surgical procedure, giving satisfactory results.

322-324 Brotherhood Block.

ABSTRACT OF DISCUSSION ON PAPERS BY DR. GIFFORD AND DR. SPAKE

DR. CHARLES T. PORTER, Boston: I differ somewhat with Dr. Gifford. I agree that the ophthalmologist is necessary in the treatment of dacryocystitis. I am not so sure that he is needed in the operation. The ideal situation would be to have the treatment under the care of both an otolaryngologist and an ophthalmologist, or one trained in both specialties. The operation presented by Dr. Gifford is unnecessarily difficult and liable to postoperative complications.

Some of the failures that Dr. Gifford had are explained by Dr. Spake's anatomic slides, which show the ethmoid labyrinth extending so far forward as to overlap and block the small opening used in this operation. I am much interested in the procedure. I have not tried it. I shall, some time, just to see whether I can do it. I should like to have Dr. Gifford explain how he expects the opening to heal and the sac to be kept patent by the surgeon's simply pushing the probe through this small opening.

I have listened with great interest to Dr. Spake, and for the most part I agree with him. It was my good fortune to have worked with Dr. Mosher in the development of the Toti-Mosher operation. I made one or two suggestions, with respect to straightening and shortening the incision and the method of closure (in two layers). These changes improved the cosmetic, as well as the functional, results.

I wish to emphasize the importance of a thorough examination of the nasal cavity and the sinuses for an obstructive lesion and infection. A high deviation of the septum can be taken care of at the time of operation on the sac, but infection of the sinuses should be cleared up first.

It is unnecessary to comment on the anatomy, which Dr. Spake has described so well. I agree with the main points of his diagnostic procedure for determining the site of the obstruction. I wish, however, to bring out several points.

1. A stricture or obstruction of the common punctum is not a contraindication to the Toti-Mosher operation. In cases of this type slitting the common punctum with a canaliculus knife or its excision, with subsequent probing or the use of a style, frequently yields excellent results. The operation is so universally fool proof and adaptable that fair results can be obtained with a little patience in cases in which the sac has previously been extirpated. I do not like to have the lower canaliculus slit before the operation, as the leaking which takes place always vitiates the results.

2. External fistula is amenable to treatment at the time of the Toti-Mosher operation.

3. I always remove the anterior tip of the middle turbinate and all cells and bone overlying the sac, with sufficient margin to prevent scar tissue from forming and closing over the sac. A second operation, however, can be performed with success if this happens. Some of the difficulty ophthalmologists have experienced has come from neglect of this technic, as well as from the method of merely slitting the nasal wall of the sac, instead of removing it.

DR. JOSEPH I. KEMLER, Baltimore: The etiologic factors in dacryocystitis have been well discussed, but there is another causative factor, namely a focus or foci of infection, either within or in close proximity to the nose. When I have a patient with dacryocystitis, whether there is suppuration or simply stricture of the duct with epiphora, I find out, for example, whether he has an infected tooth or empyema of the antrum, whether the focus is on the same side as the trouble with the tear duct or on the other side and whether he has infected tonsils or infection of a cavity not directly connected with the lacrimal sac and not in close proximity. If I can clean up such a focus, I know that I can cure the patient by probing and washing out the lacrimal sac once or twice, or sometimes by leaving it alone.

It is only when I cannot find a focus of infection that I have trouble in getting rid of the dacryocystitis, but generally I find a focus. I reported on the subject in the *American Journal of Ophthalmology* several years ago (**10**: 1, 1927). I have followed the practice of looking for such a focus in all cases, even in instances in which the patient has been subjected to probing and similar treatment for a long time and has been advised that nothing will help except an operation.

DR. LA VERNE B. SPAKE, Kansas City, Kan.: Dr. Kemler suggested a focal infection as the

cause of purulent dacryocystitis; such a cause is entirely probable and should be ruled out in the first examination. The rhinologist should not probe the nasolacrimal duct in the treatment of this condition except for diagnostic purposes. It is the ophthalmologist's work. Cases of purulent dacryocystitis are not common, but I recommend the Mosher operation when indicated.

DR. HAROLD GIFFORD JR., Omaha: I was taught to do the Toti-Mosher, or a somewhat similar, operation in Boston before I came back to Omaha and found Dr. Stokes doing the much simpler operation I have described here.

In our procedure there is not nearly as much bleeding. We dissect only the lower half of the sac. With the Toti-Mosher operation nearly the entire sac must be dissected. That is twice as much work. We do not pay any attention to the ethmoid cells unless they are infected. If there is empyema it is obvious, and the ethmoid cells are curetted. We found few instances of such a focus in 88 cases in which we performed the operation. How the ethmoid cells of infants produce mucocele is not clear. Infants 3 to 6 months of age certainly do not have extensive ethmoid cells; yet they may have mucocele.

This operation was devised primarily for the ophthalmologist, to make it easy for him. It may be a short cut, but I think one well worth while. Our percentage of cures is as good as that in Dr. Mosher's reported series. Not any better, but just as good, and I am sure that our operation is much simpler.

DR. H. P. MOSHER, Boston: Dr. Gifford said that his operation is one for ophthalmologists only and that it is a short cut. It is too short a cut for me. Results are what count. For example, one may consider the results of an ophthalmologist who was also trained in Boston, but trained from the standpoint of surgery of the nose and throat—Dr. H. B. Chandler. He performs the full, modified Toti-Mosher operation, the basic principle of which is sufficient destruction of the ethmoid cells so that there will be no recurrence of granulation tissue which can block off the opening into the tear sac. In his first series he had cures in 75 per cent of cases. He modified the Toti-Mosher operation by using a T-shaped incision and suturing one flap anteriorly and one flap posteriorly. He now maintains that he obtains a cure in 100 per cent of cases. I doubt that; it is too good to be true, but the rate is far better than the proportion of 68 cases of cure in Dr. Gifford's series of 85 cases.

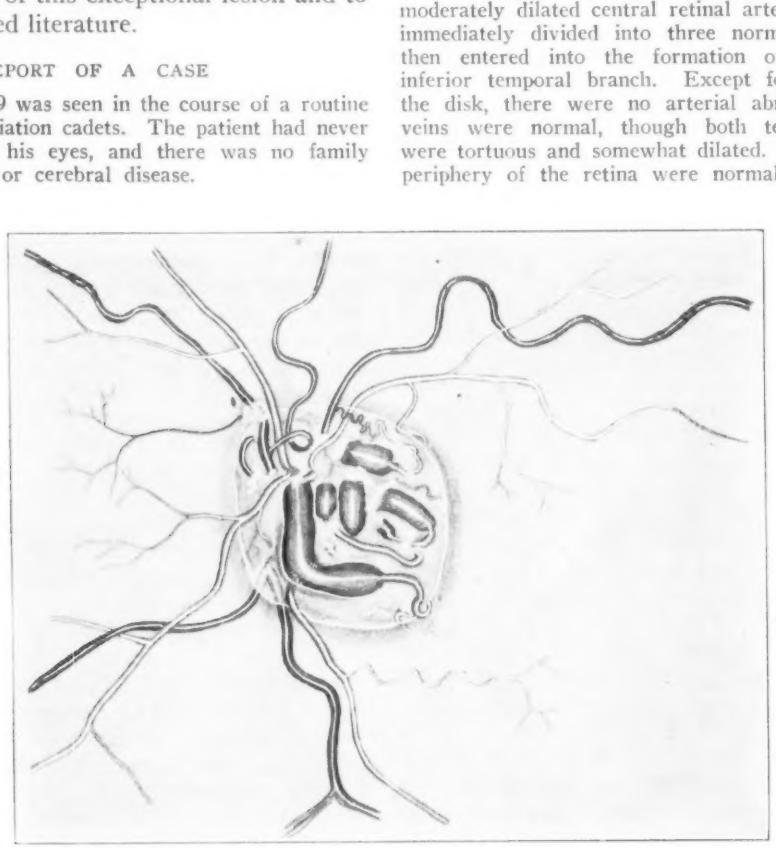
CONGENITAL ARTERIAL ANEURYSM AT THE PAPILLA

CAPTAIN FREDERICK H. THEODORE AND CAPTAIN WILLIAM H. BONSER
MEDICAL CORPS, ARMY OF THE UNITED STATES

While all types of aneurysm of the larger branches of the central retinal artery are extremely rare, the occurrence of an uncomplicated, asymptomatic arterial aneurysm of congenital origin is unique. It is felt worth while, therefore, to report a case of this exceptional lesion and to review the related literature.

REPORT OF A CASE

A youth aged 19 was seen in the course of a routine examination of aviation cadets. The patient had never had trouble with his eyes, and there was no family history of ocular or cerebral disease.



Fundus, showing arterial aneurysm of congenital origin.

Vision in each eye was 20/15 (6/5). Ocular examination revealed nothing remarkable except for the fundus of the left eye. This presented a most unusual picture (figure). The optic disk was almost entirely obscured by a serpentine aneurysmal dilatation of the inferior temporal artery, which occupied the temporal seven eighths of the nerve head. The aneurysm consisted of sausage-like, saccular coils of artery, dilated to at least four times the normal diameter, which were connected by narrow, vermiform portions and contained blood. Moderately dense glial tissue covered all but the summits of the larger dilatations, filled the interstices between the coils and extended beyond the limits of the aneurysm to obscure completely the margins of the nerve head. The arterial loops of the aneurysm projected in various planes into and out of the glial tissue, producing an elevation of 4 D. The aneurysm did not pulsate, but carotid pressure caused it to collapse partially. From the inferior portion emerged a normal artery to supply the inferior temporal quadrant of the retina. Near the nasal margin of the disk, at 10 o'clock, one could recognize the moderately dilated central retinal artery, which almost immediately divided into three normal branches and then entered into the formation of the anomalous inferior temporal branch. Except for the region of the disk, there were no arterial abnormalities. The veins were normal, though both temporal branches were tortuous and somewhat dilated. The macula and periphery of the retina were normal; there were no hemorrhages or exudates. The visual fields were normal. General physical and neurologic examinations revealed no abnormalities. There was no hypertension.

REVIEW OF LITERATURE

All types of aneurysm of the retinal vessels are exceedingly rare. The aneurysms described up to the present may be divided into three categories: first, isolated aneurysm of the central retinal artery or its first branches on the papilla, which is the rarest type of all; second, multiple miliary aneurysms of the smaller retinal arteries, which are much more common, but still a curi-

osity, and, third, arteriovenous aneurysm, which is also rare.

Aneurysm of the Larger Branches of the Central Retinal Artery.—Three cases of aneurysm of the central retinal artery were described before the advent of the ophthalmoscope; because of the absence of examinations of the fundus and the lack of modern clinical records, these cases are mentioned only for their historical interest. The first case was that of Graefe,[1] who, in 1808, described an aneurysm occurring in the optic nerve associated with what he called varices and telangiectases of the retina, discovered at autopsy in an elderly woman who had died of cerebral arteriosclerosis. She had had severe pulsating pain deep in the orbit, which decreased as amaurosis supervened. In 2 additional cases, 1 reported by Schmiedler [2] and the other by Scultet,[3] in 1818, the lesions likewise were discovered post mortem.

The first case in which the diagnosis was made on the basis of the ophthalmoscopic findings was that of Sous,[4] who, in 1865, reported a pulsating retinal aneurysm in a woman aged 64 with cardiovascular disease. She experienced greatly diminished vision and occasional pain in the eye. The aneurysm occupied the upper two thirds of the papilla. At the upper margin of the disk the mass ended abruptly, and a tortuous branch of the retinal artery appeared. The retinal arteries were extremely narrowed, a change indicating pronounced vascular alterations. Because of the lack of structure of the mass Sous concluded that it was a pulsating arterial tumor.

Raehlmann, in 1889 [5] and again in 1902,[6] in a report of his studies on retinal vascular sclerosis, described a number of small grapelike or spindle-shaped aneurysmal dilatations of the larger retinal arteries occurring in persons with arteriosclerosis, hypertension, cardiac disease and nephritis. In his cases there were poor vision, notable venous changes and retinal hemorrhages as well. In only 1 case was pulsation noted. Here, however, as a result of aortic insufficiency, the entire arterial tree pulsated. The author mentioned other small aneurysms occurring in cases of syphilis. In only a few of his cases were the arteries on the disk involved, and in none did the aneurysms resemble either in size or in type the condition in the case reported here.

In 1875 Mannhardt [7] described a small pulsating mass on the optic disk which he decided was a traumatic aneurysm because of a history of indirect ocular trauma and the coexistence of a choroidal rupture.

In 1891 Oeller [8] described a small aneurysm on the optic disk occurring in an arterial branch which supplied the macular region. The patient exhibited recurring central scotomas in each eye, which eventually resulted in bilateral optic nerve atrophy. In addition, peripheral neurologic disturbances occurred. Oeller hypothesized that multiple small aneurysms of the brain accounted for the visual and neurologic changes, as had been described by Liouville (next section). Any one reading the article at the present time would be more inclined to make the diagnosis of multiple sclerosis with an incidental small aneurysm of the retina.

A case presented briefly by Gertz [9] at a meeting of a society in Copenhagen, in 1916, appears to resemble our case more closely than any other. In a woman aged 21 the left inferior temporal artery became greatly enlarged shortly after it left the disk. Running toward the macula in great curves and twists, the vessel finally returned to the nerve head, where it disappeared under the source of the vessels, only to emerge again as a normal inferior temporal artery. Vision was 5/36. Gertz concluded that the aneurysm was probably congenital in origin. Unfortunately, the case is described in one short paragraph, and no illustration or discussion is included.

Miliary Aneurysms of the Retina.—Miliary aneurysms of the smaller retinal arteries are of two types: The first, or senile, type presents an uncomplicated ophthalmoscopic picture and is usually observed in elderly people with generalized arteriosclerotic changes. In some instances, aneurysms of the cerebral vessels have been associated with such miliary aneurysms, as was pointed out by Liouville,[10] who, in 1870, reported

1. Graefe, C. F.: Angiectasie, Leipzig, K. F. Köhler, 1808, p. 32.

2. Cited by Leber,[12] p. 20.

3. Cited by Leber.[2]

4. Sous, G.: De l'aneurysme de l'artère centrale de la rétine, Ann. d'ocul. **53**:241, 1865.

5. Raehlmann, E.: Ueber ein pulsirendes Dehnungsaneurysma der Arteria centralis retinae, Klin. Monatsbl. f. Augenh. **27**:203, 1889.

6. Raehlmann, E.: Ueber die ophthalmoscopische Diagnose sclerotischer Erkrankungen der Netzhautgefässe, Ztschr. f. Augenh. **7**:425, 1902.

7. Mannhardt, F.: Ruptur der Chorioidea, Klin. Monatsbl. f. Augenh. **13**:132, 1875.

8. Oeller, J.: Ein miliares Aneurysma einer Maculararterie, Arch. f. Augenh. **22**:68, 1891.

9. Gertz, O.: Demonstration af 2 patienter med karanomali i øjegrunden, Hospitalstid. **61**:283, 1918.

10. Liouville, H.: Note sur la coexistence d'altérations anévrysmales dans la rétine avec des anévrysmes des petites artères dans l'encéphale, Gaz. d'hop. **43**: 141, 1870.

autopsy observations on elderly patients who had died of cerebral accidents. Multiple miliary aneurysms of the arteries of the brain, meninges and retina were noted. In 1881 Litten [11] described similar lesions in the retina of a man aged 56 who had died of subarachnoid hemorrhage.

The second type of miliary aneurysm of the retina is of an entirely different nature. Although noted much earlier, it was first described as a clinical entity by Leber.[12] Only young persons, almost always males, are affected. The aneurysms are associated with exudative degenerative changes in the retina resembling retinitis circinata. With rare exceptions, only one eye is involved, with notable visual impairment. Duke-Elder [13] concluded that the condition is a type of angiomatosis and is neoplastic. While rather rare, a number of cases have been reported; we encountered 2 of them in the routine examination of 190,000 men.[14]

Arteriovenous Aneurysm.—Leber,[15] following Virchow's nomenclature, first applied the term "aneurysma racemosum arterio-venosum" to the rare and striking anomaly of an arteriovenous aneurysm involving the main branches of the retinal vessels. Subsequent authors have accepted the name "racemose aneurysm"; and the French and Italians have used it interchangeably with the term "cirsoid aneurysm." This has caused some confusion. In this condition, one or more greatly dilated and tortuous arteries arise from the optic disk and proceed peripherally for a variable distance, only to loop about and return to the nerve head as a dilated vein. The transition from artery to vein is gradual. In cases of more severe type all the arteries are involved in an anastomotic maze resembling a medusa head. All the reported cases occurred in young persons, except the case of Ehlers,[16] whose patient was 65 but probably had had the condition all her life. In some cases vision is fairly good; in others there is considerable impairment.

Recent articles concerned with racemose aneurysm, not mentioned in any of the reference textbooks, have been published by Stokes,[17] in 1934; by Bonnet, Dechaume and Blanc,[18] in 1937, and by Mozzetti,[19] in 1939. Perhaps the most important contribution is the paper of Bonnet and his co-workers. They presented 2 cases, in each of which a racemose arteriovenous aneurysm of the retina was associated with a cutaneous racemose arteriovenous aneurysm of the face. There was suggestive evidence, both clinical and roentgenographic, of a similar aneurysm of the meninges, with resulting serious neurologic accidents. The conclusions of the authors are of considerable interest. They pointed out that the simultaneous occurrence of aneurysms in the retina, the face and the brain or the meninges appears to constitute a symptom complex similar to such recognized syndromes as (1) angiomatosis retinae associated with angiomatous cysts of the brain; (2) nevi of the face associated with glaucoma, and (3) nevi of the face associated with cerebral angiomas. They stated that while the aneurysm may be observed in only one of the three sites, the triad should be borne in mind in cases of an obscure cerebral condition. When poor vision occurs, it is usually indicative of cerebral involvement. In contradistinction to the angiomas of von Hippel-Lindau disease, which they asserted have a neoplastic evolution and are made up of angioplastic capillary elements, they concluded that racemose aneurysm is congenital and involves arterioles and venules only, with no neoplastic characteristics. If further pathologic reports confirm the observations of Bonnet, Dechaume and Blanc, these authors should be given credit for the presentation of a new syndrome. While these observations are suggestive, final judgment must be reserved until definite proof of similar aneurysms of the brain is forthcoming.

Mozzetti's unusual case appears to offer corroboration of this concept. A boy aged 16 had bilateral arteriovenous racemose aneurysm of the retina, together with an "angiomatous" cyst of the neck and hemiplegia.

11. Litten, M.: Apoplexie des Gehirns und der Retina bedingt durch miliare Aneurysmen, Berl. klin. Wchnschr. **18**:25, 1881.

12. Leber, T.: Die Krankheiten der Netzhaut, in Graefe, A., and Saemisch, T.: Handbuch der gesamten Augenheilkunde, ed. 2, Leipzig, Wilhelm Engelmann, 1915, vol. 7, p. 27.

13. Duke-Elder, W. S.: Text-Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1941, vol. 3, p. 2617.

14. Theodore, F. H.; Johnson, R. M.; Miles, N. E., and Bonser, W. H.: Causes of Impaired Vision in Recently Inducted Soldiers, Arch. Ophth. **31**:399 (May) 1944.

15. Leber,[12] p. 37.

16. Ehlers, H.: Aneurysma Racemosum Arteriovenosum Retinae, Acta ophth. **2**:374, 1924-1925.

17. Stokes, W. H.: Racemose Arteriovenous Aneurysm of the Retina, Arch. Ophth. **11**:956 (June) 1934.

18. Bonnet, P.; Dechaume, J., and Blanc, E.: L'anévrysme cirsoïde de la rétine (Anevrysme racémeux). Ses relations avec l'anévrysme cirsoïde de la face et avec l'anévrysme cirsoïde du cerveau, J. de méd. de Lyon **18**: 165, 1937.

19. Mozzetti, M.: Sulle anomalie dei vasi retinici ed in particular modo sull'aneurisma cirsoide della retina, Boll. d'ocul. **17**:455, 1939.

COMMENT

The new case presented here is unusual, apparently unlike anything hitherto reported, except possibly for Gertz's case. We noted a serpentine, convoluted and greatly dilated portion of an artery on the optic disk. The artery became normal again as it entered the retina. The anomaly must be considered an aneurysm because of its saccular dilatations and definite arterial structure. It had none of the characteristics of an angioma. Essentially, it was not a tumor, although a certain amount of hyperplasia may be assumed from its appearance. Embedded in a heavy glial mantle, with no evidence of recent changes in pressure, it was undoubtedly of congenital origin, although a certain amount of growth during life must be hypothesized. One might even hazard a guess that the anomaly began at the 100 mm. stage of embryonic life, when the retinal circulation evolved from the hyaloid artery in the prepapillary cone of Bergmeister.

The absence of pulsation does not argue against the diagnosis of aneurysm. In most of the cases in which the lesion was reported to be an aneurysm pulsations were not observed. For arterial pulsations to be visible on ophthalmoscopic examination it is necessary that the pulse be of large amplitude or that the arterial and intraocular pressures approximate each other. As one might expect, aneurysm of the order encountered in the retinal arteries does not of itself alter pressure relations enough to produce pulsation unless other factors operate. The pulsating aneurysm encountered by Raehlmann occurred in a case of aortic insufficiency, in which the entire vascular bed pulsated as well.

Because of its convoluted, serpentine pattern, the aneurysm should be classified as cirsoid, as defined both by Kaufmann [20] and by Aschoff.[21] Unfortunately, a great deal of confusion and contradiction appears in the use of the terms "cirsoid" and "racemose" aneurysm. As already indicated, these names have been employed interchangeably by some authors to describe the arteriovenous type of aneurysm; still other authors have applied the term to a tumor more properly known as angioma arteriale racemosum, which is progressive, invasive and erosive.[22] These properties certainly did not characterize the present lesion. That congenital aneurysms verge somewhat on neoplasms is likely, and it is obvious that they must result from different stimuli than the aneurysms produced by trauma or degenerative vascular disease, which, with the possible exception of the aneurysm in Gertz's insufficiently described case, were the only types of purely arterial aneurysm heretofore reported. In fact, the aneurysm we have described resembled some of the arteriovenous racemose aneurysms previously mentioned to a greater extent than any other form except that it was purely arterial and was confined to the optic disk. It is even possible that the conditions differed only in the absence of an anastomosis of the artery with a vein in embryonic life.

In this connection, the reports of Bonnet and associates and the case of Mozzetti, in which racemose arteriovenous aneurysms of the retina were presumably associated with other aneurysms of the brain, suggest that in our case, too, cerebral involvement may some day be demonstrated.

SUMMARY

A congenital arterial aneurysm of a main branch of the central retinal artery is an exceedingly rare anomaly, especially when uncomplicated by visual disturbances. An extensive review of the literature revealed no case of an exactly similar lesion. There is considerable confusion in the classification of aneurysms of the retinal vessels, and it is convenient to divide the lesions in the various cases thus far reported into three types: (1) aneurysms of the larger branches of the central retinal artery, which all appear to have occurred secondary to vascular disease or trauma, and which are generally associated with visual impairment; (2) miliary aneurysms, which are of two types, one essentially a senile degenerative phenomenon, the other neoplastic, and (3) arteriovenous aneurysms, essentially congenital in origin and often without any associated visual disturbances. The third category is of additional interest in that recent articles have called attention to the association of this striking anomaly with similar arteriovenous aneurysms in the skin and the brain. The aneurysm in the case reported here corresponded to none of these types but resembled the third in several important details, although the aneurysm was entirely arterial.

20. Kaufmann, E.: Pathology, translated by S. P. Reimann, Philadelphia, P. Blakiston's Son & Co., 1929, vol. 1, p. 134.

21. Aschoff, L.: Pathologische Anatomie, ed. 6, Jena, Gustav Fischer, 1923, vol. 2, p. 81.

22. Ewing, J.: Neoplastic Diseases, ed. 4, Philadelphia, W. B. Saunders Company, 1940, p. 261.

Clinical Notes

OPERATION ON THE PUNCTUM LACRIMALE

Indications

LOUIS J. HENDRICKS, M.D., CINCINNATI

At the Ninety-Fourth Annual Session of the American Medical Association two papers [1] were read on the relief and cure of dacryocystitis by means of a window cut into the lacrimal bone. The ophthalmologist advocated cutting the window rather far forward, whereas the rhinologist expressed the opinion that the anterior ethmoid cells should be removed and, at the same time, the opening into the nose should be made through the lacrimal bone.

The ophthalmologist stated his belief that the operation was within the province of the ophthalmic surgeon, while the rhinologist was just as certain it was in the field of his specialty. To a man who does work in the two fields it seemed that a knowledge of both specialties would not be amiss in the proper performance of this operation.

However, I wondered why no one mentioned a time-honored procedure which in most cases will make the Toti-Mosher operation unnecessary. I refer to the old operation on the lacrimal point, with subsequent gentle probing of the lacrimal duct into the nose. For many years my associate and I used this procedure in the relief and cure of many conditions of the eyes. The operation is not a cure-all, since there are many cases in which nothing short of removal of the lacrimal sac or a window operation will give relief. But when the operation is carefully done, and is sensibly followed by gentle probing of the duct, the results are often surprising.

We have found that slitting and probing of the lacrimal duct helps in relief of congenital stenosis of the duct in infants, acute and chronic dacryocystitis, chronic catarrhal conjunctivitis (not accompanied by destruction of bone or scarring, as in saddle nose), catarrhal ulcer of the cornea, senile spastic ectropion and entropion and that annoying epiphora so often seen in older people. The canaliculi are slit with the use of anesthesia induced with cocaine, butacaine or tetracaine, and the procedure is seldom painful if a few drops of any of these agents are instilled before the dilating probe is gradually twisted into the sac. A probe slightly larger than a blunt-pointed darning needle is of the ideal thickness. Most dilating probes do not taper gradually enough and are not long enough to enter the sac. The probe should enter the sac because the narrowest portion of the canaliculus is usually just at the point where it enters the sac. I choose the lower canaliculus because I have personally found it easier to work through this punctum and because the results seem better than when the upper canaliculus is used.

After the probe has been passed into the sac, the canaliculus is slit with a probe-pointed canaliculus knife. It must be sufficiently sharp so that with little pressure it cuts the skin as the handle is elevated. The actual incision should be slightly on the conjunctival side of the lid, never toward the cutaneous side. Between one-third and one-half the distance between the puncta and the inner canthus is incised.

At this first operation no more is done than to stop the bleeding. Then, two days later, the same incision is kept open by running the small end of the probe along the incision and along the floor of the canal. At the same time the sac is reentered, and, if possible, a no. 1 probe is passed down the duct into the nose.

The incision must be kept open every few days until the edges are healed. At each visit the duct is probed all the way into the nose. Larger and larger probes can be used, provided they can be passed without much pressure or pain. There does not seem to be any necessity for using an instrument larger than a no. 5 or no. 6 Bowman probe.

I wish to repeat that this operation, and the gradual, gentle probing which follows it, is not a cure-all, but it deserves a fair trial in treatment of conditions which I have mentioned. Some authors have objected to the operation because it has been said to be disfiguring, but this can hardly be true if only one third of the canal is opened and the incision is made slightly on the conjunctival side of the lid. Others have said that it could not easily be followed by the Toti-Mosher operation, but, again, I believe that if only the outer third of the canal is slit a window can be successfully made in the sac and the bone.

809 Carew Tower.

1. Gifford, H.: Dacryocystitis, Arch. Ophth., this issue, p. 485. Spake, L. V. B.: Chronic Dacryocystitis: Treatment from the Rhinologic Point of View, ibid., this issue, p. 488.

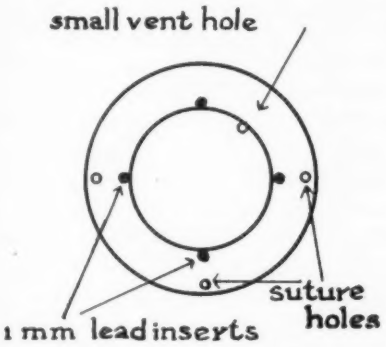
SHELL FOR ROENTGENOGRAPHIC LOCALIZATION OF INTRAOCULAR FOREIGN BODIES

Modification of Comberg's Contact Lens

HARVEY E. THORPE, M.D., PITTSBURGH

Comberg's roentgenographic method of localization of intraocular foreign bodies is extremely simple and accurate.

It consists of (1) placing a contact lens containing four lead markers on the cornea and (2) then taking a lateral and a posteroanterior view with the patient in the chin-nose position. From these two views all measurements can be taken and plotted on a sectional and an anterior view of the globe. The film measurements have to be reduced by a correctional factor of 10 per cent, this small distortion being due to the distance between the object and the film.



New shell for roentgenographic localization of intraocular foreign bodies.

The Comberg contact lens has three inherent faults.

1. It is occasionally pushed downward by the upper lid, so that the contact lens and the visual axis are not coaxial.

2. Sometimes suction develops between the localizing (contact lens) shell and the eyeball, causing difficulty in removal of the shell.

3. It is fragile, being made of glass.

In 1941 a plastic contact lens was drilled, and four metallic dots were placed at 12, 3, 6 and 9 o'clock at the limbus. This obviated objection 3.

Recently three small holes were drilled near the scleral border of the localizing shell at 3, 6 and 9 o'clock, so that scleral sutures could anchor the localizing shell to the globe. A small vent hole was drilled just inside the limbus to permit easy removal of the localizing contact lens from a traumatized globe. The figure shows the position of the holes.

If one wishes to visualize the anterior segment by stereoscopy, the glass contact lens, drilled as illustrated, is to be preferred to the plastic shell, which is nonopaque to roentgen rays.

Read before the Section on Ophthalmology at the Ninety-Fourth Annual Session of the American Medical Association, Chicago, June 15, 1944.

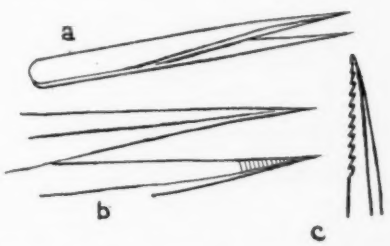
A NEW CORNEAL SPLINTER FORCEPS

HARVEY E. THORPE, M.D., PITTSBURGH

Wooden splinters and thorns wedged into the cornea are extremely difficult of removal with the usual spud. Moreover, deeply penetrating splinters and pieces of metal, such as brass lying between corneal lamellas, present additional difficulties. A modification of a fine watchmaker's tweezers proved especially helpful in removal of such splinters of wood, thorn or brass.

As the figure shows, the sharply pointed tweezer blades have several parallel grooves filed on their opposing inner surfaces. These grooves slant backward, so that backward pointing steel ridges give a viselike hold on the object grasped whether it is smooth or rough.

500 Penn Avenue.



(*a*) General shape of the forceps; (*b*) a somewhat enlarged view, showing the parallel grooves on the inner surface, and (*c*) a magnified lateral view of the tweezer blade.

Read before the Section on Ophthalmology at the Ninety-Fourth Annual Session of the American Medical Association, Chicago, June 15, 1944.

Ophthalmologic Reviews

EDITED BY DR. FRANCIS HEED ADLER

THE CRYSTALLINE LENS IN DIABETES MELLITUS

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MEDICAL CORPS, ARMY OF THE UNITED STATES

The first mention of an ocular derangement associated with diabetes mellitus apparently was made by Blancard in 1688; he described the case of a diabetic girl who became blind just before death. However, destruction of the optic nerve, not lenticular alterations, caused the blindness. Saunders, who in 1798 described the onset of bilateral cataract in a diabetic person, was the first to draw attention to the relation of the two conditions. Subsequently sporadic reports appeared so that by 1854, when His described the microscopic pathology of diabetic cataract, more than a dozen authors had already observed this type (Destouches; Jahn; Berndt; Unger; Liman; Himly; T. G. G. Benedict; Oppolzer; His; Lohmeyer; Bouchardat, 1846 and 1852; Ruete, 1843 and 1854, and Mackenzie).

Disturbances of the eye in diabetic patients are extremely common. Since ocular disturbances are most frequently seen in persons over 40 years of age, senile changes must always be considered as an etiologic factor. Nevertheless, there appears to be a significant relation between the incidence of ocular complications and diabetes. The lens and the retina are the organs most frequently affected, well over half of all the cases of visual disturbances being attributable to changes in these structures.

Two types of abnormalities of the lens appear: (1) transitory refractive changes and (2) diabetic cataract.

TRANSITORY REFRACTIVE CHANGES

Refractive changes in the lens were not reported in the literature until the middle of the nineteenth century. Desmarres (1858) stated that when the ability to read diminishes and the need for stronger glasses increases a refractive change is indicated. Rosenstein found that the eyes of a 35 year old diabetic patient were "presbyopic." The patient's ability to read improved with the wearing of a strong convex lens. In the same year von Graefe reported weakness of accommodation in diabetic persons in whom refractive changes had taken place. Horner (1873) described increased hyperopia in such patients. Hirschberg (1890), who first recognized myopia accompanying diabetes, stressed the fact that the onset of myopia in adults should arouse suspicion of diabetes. Since then, a voluminous literature concerning the clinical course and pathogenesis of this complication has appeared.

The transitory refractive changes associated with diabetes mellitus are equally distributed throughout all age groups (Granström). Occurring only in cases of fresh diabetes with a high level of blood sugar and with glycosuria, diabetic myopia is less common and of lesser degree than diabetic hyperopia. The hyperopia always follows the myopia before a return to the normal refractive state can occur; that is, the affected patient's vision passes from myopia to hyperopia before returning to a normal condition. Hyperopia, on the other hand, may arise without preceding myopia. It is only when the patient's general condition begins to improve that the refractive changes of hyperopia appear. Zentmayer pointed out that in his cases, and in other reported cases, failure in vision occurs when the percentage of sugar in the urine begins to decrease. Hagen, Duke-Elder (1925), S. F. Gifford (1928), Granström and others have confirmed these observations. In sharp contrast to the rapidity of development of the hyperopia, the regression to a normal state is gradual, generally requiring two to four weeks. Weakness of accommodation accompanies hyperopia in persons under 30 years of age; rarely is this combination encountered in older persons.

The theories proposed for the pathogenesis of the refractive changes associated with diabetes mellitus fall into two groups: anatomic and physicochemical.

ANATOMIC THEORIES

Loss of Accommodation.—Von Graefe expressed the belief that paresis of accommodation allows a latent hypermetropia to become manifest.

The objections to this theory are that the range of accommodation in persons with cataract complicating diabetes is frequently normal and that it does not explain the instances in which

myopia occurs. Hudelo's modification of this hypothesis is based on the fact that the ciliary muscle consists of two antagonistic portions—the sphincter and the tensor choroidea (Brücke's muscle). He proposed a new theory of accommodation, the physiologic basis for which lay in a dual nerve supply. The sphincter muscle is innervated by the oculomotor nerve and the radiating fibers by the sympathetic nerve. In accordance with the muscle paralyzed, hyperopia or myopia develops. A schematic presentation of his theory follows:

> Paralysis of sphincter muscle + integrity of tensor choroidea = slight hypermetropia.
>
> Paralysis of tensor choroidea + integrity of the sphincter muscle = moderate myopia.
>
> Paralysis of both muscles = loss of accommodation. The refraction remains in its original state.
>
> Paralysis of pars ciliaris = astigmatism.

Changes in Extraocular Muscles.—It is possible that alterations in the extraocular muscles may be a factor in refractive changes associated with diabetes. Thus, according to Duke-Elder, who cited a case reported by Risley, the hypermetropia appeared and disappeared in proportion to the degree of esophoria.

Swelling of the Ciliary Processes.—Scalinci stated the belief that the swelling of the ciliary processes occurring with diabetes mellitus compressed the lens at the equator and caused the refractive change. Elschnig (1929) expressed the opinion that a similar swelling caused slackening of the zonular fibers, producing myopia, while shrinking of the ciliary epithelium increased the traction of the zonular fibers, producing hyperopia.

Elongation of the Globe.—De Schweinitz, basing his opinion on the observation of a woman aged 20 with diabetes and choroidal edema, expressed agreement with the theory proposed by Knies that myopia associated with diabetes is axial in type and is produced by stretching of the sclera by the underlying edema.

Shrinkage of the Globe.—Shrinkage of the bulb, proposed by Horner to explain the hyperopia accompanying diabetes, is based on the well known presence of a soft eyeball in cases of diabetic coma. This theory has repeatedly been disproved by the fact that the corneal measurements and the intraocular tension remain normal in persons with refractive changes complicating diabetes.

PHYSICOCHEMICAL ALTERATIONS

Changes of a physicochemical nature which produce alterations in the ocular media are the basis of the more prevalent theories.

Changes in the Index of Refraction of the Aqueous Humor.—Schapringer and Appenzeller independently assumed that the increased refractive index of the aqueous humor, caused by a greater sugar content, plays an important part in the myopia of diabetes. Appenzeller noted in a case of diabetes that myopia persisted as long as glycosuria was present. According to Hagen and Duke-Elder (1925), it is improbable that the aqueous humor ever has a sugar concentration so high as to cause an appreciable change in the refractive index. Further, if the sugar in the aqueous humor should reach an extremely high concentration, it would result in cloudiness of the lens. Hagen computed that the dextrose content of the aqueous in the eye of a diabetic patient with myopia was about 1.3 per cent. The index of refraction of this solution is higher than the normal.

Changes in the Index of Refraction of the Vitreous.—De Wecker and Landolt explained transitory refractive changes in cases of diabetes by the increased refractive index of the vitreous. They wrote: "L'augmentation de l'indice de refraction du corps vitre doit faire diminuer également la refraction de l'oeil." (Increase of the refractive index of the vitreous body should also diminish that of the eye.) Adler expressed the belief that the pathogenesis of the refractive changes may be based on the difference in the response of the aqueous and that of the vitreous to the level of sugar in the blood. The former parallels the sugar content of the blood; the latter lags behind. On the basis of Adler's experiments, Cowan suggested:

> . . . if the refractive index of either the aqueous or vitreous alone changed [in diabetes] a much smaller change would account for the development of a considerable ametropia. Thus, if the sugar concentration of the aqueous increased while that of the vitreous remained constant, the resulting difference of the refractive indices in the two media would account for a considerable myopia. On the other hand, if the vitreous sugar were high and the aqueous alone dropped, a considerable hyperopia would be developed.

Changes in the Index of Refraction of the Lens.—Most modern investigators have stated the belief that refractive changes associated with diabetes are dependent on an alteration in the crystalline lens. Duke-Elder, especially, stressed the importance of the osmotic relation between the aqueous humor and the lens. Using Hess's technic of illumination of the lens, Woelfflin found, instead of the four images normally seen in persons over 25 years of age, only two distinct images, an observation demonstrating that when refractive changes occur in the course of diabetes, differences between the nucleus and the cortex of the lens decrease. This observation

was confirmed by Grönholm (1911) and Enroth. According to Gullstrand, the myopia and hyperopia of diabetes may be explained by an alteration between the surfaces of equal indexes of refraction. Rönne assumed that hyperopia results from a negative intracapsular accommodation, while Lundsgaard and Spalding stated that the flattening of the surface of the lens is an important factor in these changes. Granström, in stigmatoscopic examinations, found that only the axial portion of the optic system was affected, while the peripheral part remained unchanged. The refractive state must arise as a result of a change in the lens, external or intracapsular. Mechanical and anatomic considerations eliminate the former mechanism. Granström concluded therefore that since the peripheral portion of the refractive system is unaffected, the refractive changes of diabetes must depend on a change in the refractive index of the nucleus of the lens. A decrease or an increase is reflected by hyperopia or myopia respectively.

The changes in the refractive index of the lens depend on the mechanism termed "dry salt retention" by Klein, in his work on the pathogenesis of latent edema in severe diabetes. The tissues retain the salt in cases of severe diabetes, with a high sugar concentration of the blood and urine. That salt retention occurs was confirmed by Kühn and Witscher, who demonstrated hypochloruria in patients with severe diabetes. With the decrease in the sugar content of the blood and urine, the tissues, because of their high salt content, attract water.

The lens, together with the other tissues, participates in the process of salt retention and secondary water uptake. During the time that a high blood sugar level and glycosuria exist, dry salt retention in the lens produces an increased refractive index (myopia). With improvement in the general condition (decrease in the amount of sugar in the blood and in the urine), the osmotic pressure of the aqueous becomes less than that of the lens. As a result of the osmotic difference, water enters the lens, with reduction of its index of refraction (hyperopia).

CATARACT AND DIABETES MELLITUS

Etiologic Factors.—The statistics on diabetic cataract are extremely difficult to interpret, as the values are vitiated by the looseness with which the term is applied. Little uniformity can be found in the literature either for the population sampled or for the criteria adopted. Some reports are based on detection on examination with the slit lamp of an opacity under conditions of complete mydriasis, while others depend only on the results of cursory ophthalmoscopic examination. The age, sex, race, occupation, number of cases, duration of diabetes and economic status are all variables which affect the data obtained. It is not surprising, therefore, that diabetic cataract has been reported variously as nonexistent, rare and common.

Age.—Much of the confusion in designation of a cataract as of diabetic origin could be avoided by a more careful classification on the basis of the age of the patient and the findings with the slit lamp. The term "diabetic cataract" has too frequently been used to indicate any cataract occurring in a diabetic person, whether or not the diabetic condition had any causal relation to the opacity. More accurately, it should refer to the characteristic cataract almost invariably found in young persons with diabetes (France). In older diabetic patients the cataract becomes indistinguishable from senile cataract (Knapp, Becker, Deutschmann and others). Although several hundred cases of diabetic cataract have been reported, the condition is relatively rare. Schnyder (1923), who was one of the first to utilize modern methods in his examination, could find only 1 case in 59 cases of diabetes in which the opacity could be designated as true cataracta diabetica. Gradle (1926) described 3 cases in 76 cases of diabetes; Jofe, 1 case in 60 cases of diabetes; Braun (1937), 4 cases in over 700 cases of diabetes; Gray (1931), 2 cases in 500 cases of diabetes, and Landabure and Lagleyze, only 3 typical cases of cataract in 1,091 cases of diabetes.

O'Brien, Molsberry and Allen, after a careful study of the problem, concluded that cataract was not as infrequent in diabetic children as the reports just cited indicated. They found characteristic opacities in 20 of 126 young diabetic patients, ranging from 3 to 33 years of age. All but 2 of these 20 patients had severe, prolonged, poorly controlled diabetes.

Although it usually appears during adolescence or early adult life, diabetic cataract may also occur in infancy. Chapiro, in 1914, reported a case of this condition in an infant and stated that it was the tenth such case to be recorded. The youngest patient whose case has been reported in the literature was an 11 month old infant (Major and Curran).

At the other extreme was a patient aged 49 whose case Elschnig (1924) reported as one of true cataracta diabetica. Braun (1937) questioned whether this case could be validly included. The age of 40 is usually taken as the maximum at which diabetic cataract can be identified with confidence. Even lower age limits

have been set by other investigators. Gray (1933) stated the belief that only cataract appearing in diabetic patients under 36 can be called true cataracta diabetica. O'Brien, Molsberry and Allen (1934) considered only patients younger than 33 in compiling their data.

Influence of Diabetes on Senile Processes in the Lens.—The relation of diabetes and cataract in older persons remains the subject of much controversy. Convincing data on this problem are extremely difficult to obtain owing to complications introduced by senility itself. It is generally conceded that morphologically typical senile cataract cannot be differentiated from cataract seen in old diabetic patients. Diabetic cataract can be distinguished from senile cataract histologically only by the pigmentary differences in the eye already described (Kamocki, 1887). In a diabetic person the pigment throughout the eye is more fragile and easily dislodged.

Since no clearcut histologic distinction can be found in the lens itself, evidence for a diabetic origin is confined largely to statistical analyses of appropriate samples of the population, although such sampling is admittedly fallible.

Gallus (1919) denied unequivocally the possibility that a diabetic state might contribute to production of cataract. Poulard also cautioned against the designation of every cataract occurring in a diabetic person as true diabetic cataract. Gradle stated that the incidence of cataract among diabetic persons is no greater than that among nondiabetic persons of the same age. These observations were substantiated in larger series of patients by Waite and Beetham and by Lottrup Andersen. In disagreement with these investigators, Römer stated the belief that diabetes at any age might lead to a characteristic identifiable cataract.

Intermediate between these opposing factions were the ophthalmologists who stated that diabetes might aggravate or hasten opacification of the lens initiated by senility (Shepardson and Crawford; van Heuven and Hulst; Kirwan; Duke-Elder, 1941). Since the ravages of diabetes are in many respects similar to those of age, a diabetic person might be expected to evidence signs of senility which otherwise would not appear for many years. Anthonisen tabulated the data on all patients undergoing operation for cataract and compared the incidence of cataract for diabetic persons with that anticipated from the incidence of diabetes in various age groups in Denmark. His data indicate a relation between diabetes and cataract unexplainable by age alone. Thus, in every age group from 25 to 75 years the number of diabetic persons among the patients with cataract was many times that anticipated from their distribution in the general population. In the younger groups, this was especially apparent, fifty to two hundred times the expected percentage being noted in some cases. Unfortunately, the significance of these values was weakened by a relative paucity of patients under 55 years. The relation of cataract to the duration of the diabetic condition remains unsettled. Lottrup Andersen could detect no difference in the time of appearance of cataract between patients with diabetes of two years' duration and patients with diabetes of five years' duration. Kirby (1933) contended that the duration of the disease, rather than its severity, was important, the highest percentage of cataracts occurring among patients with diabetes of long standing. His figures, however, are not convincing. Cataract was found in 40 per cent of diabetic patients who had had the disease for five years and in 62 per cent of the patients with diabetes of two years' duration.

Frequency of Hyperglycemia in Patients with Cataract.—The relation between cataract and diabetes may be proved either by showing that the incidence of cataract among diabetic persons is abnormally high or, conversely, by demonstrating that a disproportionate number of cataractous patients possess a deranged carbohydrate metabolism. The former approach has been discussed in the preceding paragraphs. The latter method has given rise to rather confusing data, which also in general support the concept that an abnormal utilization of carbohydrate may contribute to opacification of the lens. Glycosuria and, more frequently, hyperglycemia or decreased sugar tolerance have been found in cataractous persons. Some authors have attributed these indications of improper carbohydrate utilization entirely to senile changes, with no causal relation to the production of cataract. Thus, Kirby and Wiener (1933) maintained that a tendency to diabetes and cataract are but manifestations of the same functional derangement appearing in old age. Nor could Lottrup Andersen, Melhose, Piper or Aliquò-Mazzei find satisfactory evidence relating cataract to an increase in blood sugar. Injury to the pancreas may be the fundamental disturbance in production of such cataracts.

Pathogenesis of Diabetic Cataract.—Although a few authors have stated the belief that diabetic cataract is a direct manifestation of pancreatic insufficiency (Löwenstein, 1934; Schiøtz), of an endocrine imbalance (Nobecourt and Ducas) or of old age (Gallus, 1919), most authors have

attributed the opacity to an osmotic or a toxic action of the excessive dextrose and its metabolic products. Proof that diabetes may give rise to cataract is furnished by the experiments of Chaikoff and Lachman. Of 10 pancreatectomized dogs kept alive by means of insulin for two years, cataract developed in 8. On the other hand, no lenticular opacity appeared in a large number of normal dogs observed during this period. Poisoning of the lens by high concentrations of dextrose was early postulated by von Graefe and Stoeber, and later by Ascher. Such a direct action seems unlikely in view of the high concentration of sugar in the aqueous humor which would be necessary. Kirby, Estey and Wiener (1933) could detect no toxic action on isolated lens epithelium until a concentration of dextrose of 578 mg. per hundred cubic centimeters was attained, a level several times higher than that reached in the aqueous of diabetic patients. Furthermore, as Gradle (1926) pointed out, cataract may develop in a diabetic person independently of the dextrose concentration in the body fluids. A diabetic patient with pronounced hyperglycemia may have a clear lens, whereas a patient with a relatively low blood sugar may display opacity. Braun (1937) also challenged hyperglycemia as a cause, asserting that he was able to produce regression of opacities of the lens by treatment with insulin. His observations, however, lack confirmation.

That cataract may result from the direct action of dextrose on the lens thus appears improbable. The possibility cannot be ignored, however, that dextrose may indirectly mediate the lenticular changes, as shown by laboratory studies. Moderate concentrations of this sugar sensitize lens proteins, which are precipitated more readily when exposed to light. In a solution of dextrose the capsule of the lens undergoes a decrease in permeability, which becomes much more pronounced when the capsule is in contact with an equivalent concentration of galactose (Bellows and Rosner, 1938).

Osmotic differences between the lens and the aqueous humor have been postulated to explain both the refractive errors and the cataract associated with diabetes (Duke-Elder, 1925, 1926). As early as 1857, turbidities were obtained by immersing freshly removed lenses in strongly hypertonic solutions of dextrose (Kunde). Injection of dextrose subcutaneously (Kunde, Richardson), intraperitoneally (Richardson), subconjunctivally (Manca and Ovio), intraocularly (Richardson) and intravenously (Viterbi and Foà) caused opacities to appear. Bellows and Chinn (1941) were unable to produce cataract by intravenous injection of dextrose, although opacities in the lens readily developed after intravenous injections of galactose, xylose and arabinose. The high concentration of dextrose required to produce the "osmotic" cataract is probably the most formidable argument against such a mechanism in persons with diabetes. Lenses dropped in a 2 per cent solution of dextrose (Deutschmann) underwent no change in turbidity. It was necessary to immerse the lens in a 5 per cent solution of dextrose before a faint cloudiness appeared. Such a concentration is fifty times that of normal aqueous and about twenty times that in cases of severe diabetes. This was emphasized by Ask in 1915 and by many authors since. It must be remembered, however, that clinically changes in the water and salt contents occur simultaneously with increased dextrose concentrations and, further, that the derangements are maintained for much longer periods than those in the laboratory. In some instances, therefore, diabetic cataract may depend primarily on an osmotic derangement of the ocular media.

The action on the lens of increased acidity of the aqueous humor in diabetes was one of the first theories advanced to explain the opacity (Lohmeyer). It was vigorously denied by Ruete (1854), Jany, Kako, Leber, Parsons and others. More recently it has been revived in modified form by Goldschmidt and Krause (1934). Goldschmidt found that the acidity of the aqueous was high in cases of diabetes. This excessive acidity, he concluded, inhibits the various internal respiratory systems and results in lowered metabolic activity. In this connection, Cohen (1921, 1929) stated the belief that the fundamental cause of diabetic cataract was nutritional in character, the lens in a case of diabetes being unable to utilize dextrose in a normal manner. Krause (1934) postulated a local acidity of the lens which may cause death of the fibers and provide optimum conditions for proteolytic decomposition.

Probably the most unassailable of all theories is the concept that the deranged metabolism in a diabetic person gives rise to toxic products which poison the lens directly or indirectly. Kirby and associates (1933) found that acetone and betahydroxybutyric acid kill lens epithelium in vitro within forty-eight hours when their concentrations equal those occurring with severe, uncontrolled diabetes. With concentrations approximating the aqueous content in cases of mild diabetes the deleterious action was slight. Schanz demonstrated that acetone was a powerful sensitizer of the lens protein to light. A specific, but unidentified, "cytotoxin" was postulated by Mauthner and Römer. The latter investigator

claimed that substances which broke down lens tissue (lens antibodies) were found in the blood of a diabetic child. Support is lacking for such an immunologic concept.

In opposition to these reports, many investigators have shown that cataractous persons frequently possess an inadequate mechanism for utilization of sugar. The incidence of hyperglycemia and decreased tolerance for carbohydrate was much greater than could be anticipated from senile changes alone. From 40 per cent (O'Brien, 1932) to 75 per cent (Baldwin and Barthel) of persons with opacities of the lens had a fasting blood sugar level exceeding 120 mg. per hundred cubic centimeters, a value customarily taken as the maximal normal concentration. In one-half the patients (O'Brien, 1932) or more (Langdon) the sugar tolerance was subnormal. Blaisdell, Galindez, Malkin and Krassikov, Palmerini and others confirmed these observations. In a limited series of dogs, owls, parrots and monkeys with spontaneous development of cataract hyperglycemia was invariably noted, the level of sugar ranging from 148 to 271 per cent of that in the control animals (White).

Chemical Changes.—Little has been uncovered concerning the chemical changes taking place in the lens of a diabetic person. As has been discussed earlier, rapid alterations of the water and salt content of the lens appear to be responsible for the transitory refractive aberrations. Carey and Hunt compared diabetic cataracts with senile cataracts and could find no distinguishing chemical characteristic except for a low phosphorus content in the former. The significance of this observation is not known. Perhaps it is related to the decreased utilization of carbohydrate, since dextrose is dependent largely on phosphorus compounds for its metabolism. Accumulation in the lens of acid bodies or other toxic products, although occasionally assumed for theoretic purposes, has never been unequivocally demonstrated. An interesting relation between naphthalene and diabetic cataract has been pointed out by Michail and Vancea, who observed that the opacities produced in rabbits by feeding of naphthalene regressed after administration of insulin.

Many investigators believe that the lenticular changes result secondarily from injury to the ciliary epithelium or to the vascular endothelium. Disturbances in the nutrition of and removal of waste from the wound in the lens result, producing at first refractive changes and later opacities (Kamocki, 1887, 1892; Peters, 1893; Parsons; Viterbi and Foà; Elschnig, 1929). The degenerated and edematous state of the ciliary epithelium in the diabetic eye has been presented in support of this theory. Duke-Elder (1926) disputed this conclusion, contending that the edema is purely coincidental to the primary osmotic changes simultaneously taking place, and maintained that the degenerative condition of the ciliary epithelium has no causal relation to the similar condition appearing in the lens.

Histopathologic Changes.—Most authors have agreed that the histologic changes in diabetic cataract are similar to those of cortical cataracts of various causes. They stressed the extreme lenticular swelling, which may lead to disintegration of the peripheral lens fibers, in addition to swelling and fissuring of the cortex. In many instances the nucleus of the lens remains unchanged. There is irregular staining of the nuclei of the capsular epithelium; in some instances these nuclei may be absent or are replaced by vacuoles. Proliferative changes often take place in the epithelium. A characteristic histologic feature is the pronounced swelling and loosening of the uveal pigment (Kamocki, 1887). The pigment lightens in color and clumps together, while the cells become swollen and vesicular, eventually rupturing to produce cystlike spaces, formed by connecting cell membranes and visible even macroscopically. Pigmented shreds cling to the walls or are seen in the debris, together with disintegrating nuclei and fine, pale, round droplets.

Symptoms.—One of the most characteristic clinical features of diabetic cataract is the rapidity of its onset. Opacities may appear in the course of days, or even hours (Scheffels, 1898; Neuburger; Litten; Chapiro; Major and Curran; Nobecourt and Ducas; Braun, 1939). The lens may become completely opaque within forty-eight hours (Nobecourt and Ducas). The cataract is almost invariably bilateral, usually appearing in both eyes simultaneously (Nobecourt and Ducas; Zeller; Hirschberg, 1890; Lippmann).

Schnyder (1923, 1924) and Koeppe were the first to examine a diabetic cataract with the slit lamp. Schnyder found the entire visible surface of the lens involved. The opacities were small and flaky, measuring 0.04 to 0.1 mm. in diameter. Posteriorly, at the pole of the lens, these areas became confluent, forming an irregularly thickened, white superficial opacity. The superficial lens fibers became more distinct, with clefts between them containing fluid. Schnyder asserted that this involvement of the superficial lens fibers was the most characteristic change in diabetic cataract. Turbidity and vacuolation of the entire subcapsular surface of the lens could be seen. As the lens continued to swell, the subcapsular fluid raised the capsule, causing disappearance of the flaky opacities. The zones of discontinuity became less distinct and finally de-

veloped into an asbestos-like, shiny cortical opacity. At times the epithelium appeared as a pellicle, floating in the anterior subcapsular fluid. Koeppe characterized this type of cataract as consisting of localized, jagged, netlike opacities involving the anterior and the posterior cortex, frequently in conjunction with vacuolation and brilliant coloration. Goulden confirmed this description in detail. He pointed out that in diabetic cataract the typical subcapsular opacities invade the space between the capsule and the band of disjunction; in all other forms of cataract this space remains clear. Gifford (1928) stated that the "true diabetic cataract is characterized by the appearance of fluid vacuoles under the capsule of both lenses, which progress rapidly to complete opacity in periods of a few weeks to six months."

Later studies confirmed the correctness of these fundamental distinctions. Case reports were contributed by Sourasky, Badot, Kirwan, Marx, O'Donoghue, Braun (1937) and many others. Especially well documented was the report of O'Brien, Molsberry and Allen, already mentioned. The authors described two common types of cataract prevailing in diabetic children. To one type O'Brien and associates applied the term "snowflake or snowstorm cataract," since its appearance was that "of a heavy snowfall against a leaden sky." This cataract was seen in 12 of the 20 children, appearing as innumerable grayish to bluish white, flaky opacities. The opacities began in the anterior and posterior cortex immediately under the capsule and in more advanced stages occupied the entire cortex. The other common type of diabetic cataract was a saucer-shaped posterior subcapsular opacity composed of confluent granules. Iridescent crystals were present in some cases subcapsularly, and more rarely in the cortex. O'Brien described fine, granular strands extending from the equatorial zone to the central opacities. Nobecourt and Ducas, and later Bangerter, described still another form in which the flocculent changes were not confined to the subcapsular tissue but appeared throughout the substance of the lens.

Medical Treatment.—Although it is always difficult to evaluate the effect of treatment on any cataract, control of the diabetic condition seems in many cases to be distinctly beneficial. Vigorous insulin treatment may delay or arrest the process in the lens, or even in rare cases cause regression of diabetic opacities if instituted in the early stages (F. Fischer; Braun, 1937; Ascher; Wirth; Crocco; Salvati; Leyton; Burger; O'Brien and associates). Only true diabetic cataract is influenced by administration of insulin; senile cataract remains unaltered.

Of inestimably more importance than its use for the control of opacities in the lens is the administration of insulin before cataract extraction in cases of diabetes; hemorrhage and infection were common operative sequelae before the insulin era. Even in the hands of a skilful surgeon, almost half the eyes of young diabetic persons became infected after extraction of the cataractous lens (H. Gifford, 1911). Infection among other patients with cataract ranged from 1 to 6 per cent. In a series of over 2,000 cases of cataract in nondiabetic patients, Wheeler reported hemorrhage in less than 5 per cent. Among diabetic patients hemorrhage was six times as prevalent. Today, surgical intervention on the eye of a diabetic person is postponed whenever possible until dietary or insulin treatment has reduced the glycosuria and hyperglycemia. Benedict cautioned against the use of large doses of insulin prior to operation, asserting that excessive amounts may increase the tendency to hemorrhage.

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Obituaries

GEORGE SLOAN DIXON, M.D.
1853-1944

It is on the note of warm friendship that I venture to tell the story of one who was an honored and cherished landmark at the New York Eye and Ear Infirmary. As a back country youth, stemming from New Brighton, Pa., George Sloan Dixon came to the city of New York in 1876—the year of the Centennial Exposition in Philadelphia. New York had still to wait nine years before it could celebrate the completion of the Brooklyn Bridge. Up to this time George Dixon had worked in the shop of his father, who was a wheelwright. There was always a certain ruggedness about Dr. Dixon which probably harked back to his early wholesome training.

The immediate purpose of his leaving home was to study stenography and typewriting. These two arts had been so far advanced that the business world was beginning to find them of practical use. Dr. Dixon used to relate that as a form of practice he would go over to the famous Plymouth Church in Brooklyn to record Henry Ward Beecher's sermons—a most difficult task because once Dr. Beecher got started he was wont to speak at the rate of three hundred words a minute.

GEORGE SLOAN DIXON, M.D.
1853-1944

By chance, in 1882, Dr. John E. Weeks, who had just finished an internship in the New York Ophthalmic and Aural Institute, met George Dixon. An intimate friendship grew up that was

to last two and sixty years. Discovering that in time spared from stenography Dixon was examining diatomes under the microscope, Dr. Weeks persuaded him to take up the study of medicine. So it came about that this erstwhile wheelwright and stenographer turned physician.

He was graduated from Bellevue Hospital Medical College in 1885. Having served as house surgeon of the New York Orthopoedic Dispensary and Hospital, and later as assistant surgeon to the Dispensary, he started out in practice as an orthopedic surgeon. During this period, in order to widen his knowledge, he served as physician in charge of the class in general medicine at the De Milt Dispensary, a once famous charity, no longer in existence.

In 1893, aware of Dr. Dixon's bent toward microscopic work, Dr. Weeks induced him to become associated with him as assistant pathologist at the New York Eye and Ear Infirmary. Dr. Weeks, in recognition of his work on the histopathology of the eye and on bacteriology at the Knapp Hospital, had been commissioned to establish a laboratory at the Infirmary, in addition to conducting a clinic.

Dr. Dixon had now found the perfect niche in medicine for the development of his unusual talents of observation and invention. This was long before the days of full time paid men. He continued to practice orthopedics, while giving much time to the laboratory gratuitously. During this period, with unbounded capacity for work, he served as assistant to Dr. Fred Whiting, in charge of the class of otology at St. Bartholomew's night clinic—another famous charity gone out of existence. He also became associated with Dr. Gorham Bacon as assisting attending surgeon in the otologic department of Cornell University Medical College and was with him later in the department of otology at the Vanderbilt Clinic. These associations with two outstanding specialists of the time were the reward for Dr. Dixon's accomplishments in the art of photography. His lantern slides of diseases of the eye and the ear were greatly sought after by lecturers and writers and had brought him into prominence. He devoted much time in an endeavor to make photomicrographs of pathologic specimens of the eye and ear, but, finding that the vibrations at the Infirmary, even at midnight, were too great, he was compelled to give up this effort. He devised a method of photographing microscopic museum specimens of the eye mounted in glycerin jelly without showing the glass jar that contained them. His photographic work had prepared him for the greater work of his life in the field of roentgenology.

In time, Dr. Dixon was appointed pathologist to the New York Eye and Ear Infirmary and later director of the laboratory, which had come to be known as the Eno Laboratory, in honor of Dr. Henry C. Eno, one of the surgeons, through whose generosity it had been equipped.

On Nov. 24, 1904, a memorable event occurred at the Infirmary, namely, the scientific localization of a foreign body in the eye by Dr. Dixon and Dr. Weeks—in all probability for the first time on this side of the Atlantic. For this purpose they made use of a McKenzie-Davidson apparatus brought over from London by Dr. Henry D. Noyes.

It soon followed that a department of roentgenology was established, and Dr. Dixon was appointed director. The work in the laboratory grew by leaps and bounds, and the technic had become elaborate. The duties of the roentgenographic department had also become most exacting. Unable to carry the double burden, Dr. Dixon resigned the post as pathologist and devoted the rest of his life to the roentgenology of the head. For the first ten years after the opening of the department of roentgenology the work was devoted mostly to the localization and plotting of foreign bodies in the eye. It was not uncommon for Dr. Dixon and his staff to be called on to locate foreign bodies in a half-dozen patients a day. Some of these bodies had been in the eye for ten or twelve years, and one, Dr. Dixon used to recall, had been present for forty-two years.

Dr. Dixon, with a knack for improving everything that he touched, made many modifications in the technic of localizations of foreign bodies in the eye. A description of an apparatus that he designed for this purpose, still in use at the Infirmary, was published in the reports of the New York Eye and Ear Infirmary in 1906 and, again, as late as 1936, by Dr. Edward Hartmann in a journal in France. Not many years ago he designed a "holder" to fix the head while roentgenograms of the anterior segment of the eye are taken from five directions. His plottings were noted for accuracy, seldom varying more than 1 mm. from the true site. Prior to the use of roentgen rays an instrument called the sideroscope had been employed to detect the presence of steel or iron in the eye. Once the foreign particle was detected, magnets were used in attempting the extraction, and the surgeon had to trust to luck for the direction.

By 1912 the roentgenographic technic of the accessory sinuses had been well developed; but that of the mastoid process lagged behind, and what knowledge had been acquired was not ac-

cepted. After many experiments, Dr. Dixon succeeded in finding the proper position for placing of the head in order to get the correct dimensions of the temporal bone. He had now become an outstanding specialist in the roentgenology of the head, more particularly of the sinuses and of the temporal bone. Many surgeons visited the roentgenologic department to seek advice. He was noted for sound judgment. In the years of his prime he attended many meetings and was active in the discussions.

Early in the days of the pathologic laboratory a young man, Edward B. Burchill, previously an apprentice to an establishment manufacturing jewelry, was assigned to help Dr. Dixon, who was in the habit of making his own equipment. By example and word, and with infinite pains, Dr. Dixon was able to help him develop his talents, until today Dr. Burchill is noted far and wide for his scientific attainments. He proved to have a rare fusion of hands trained in mechanical dexterity and eyes keen to observe minutiae. Dr. Burchill's preparations of countless skulls demonstrated that there are innumerable variations in the anatomy of the accessory sinuses and of the temporal bone, many of which had been little known before. Dr. Dixon always said that without the study of these specimens he would have missed many things in his plates or would have been puzzled to account for many things that he did see.

On Oct. 15, 1935, after forty-two years of active and dutiful service, and in the eighty-third year of his age and the one hundred and fifteenth year of the Infirmary, Dr. Dixon resigned his post as director of the department of roentgenology and was appointed consulting surgeon. His time at the Infirmary had bridged a vast change in the specialties of ophthalmology and otology. Particularly was this true in regard to the teaching of the pathology of the eye and ear, the lack of knowledge of which had been appalling. The subject was considered a waste of time. Dr. Dixon lived to see its study a major requirement at the Infirmary. Owing to the enforced use of goggles by workmen and other preventive measures, as the decades passed, he was called on less and less to take roentgenograms of the eye. The reverse had happened—the ophthalmic work had grown lighter, and the work with the sinuses and the temporal bone was heavier.

On Jan. 18, 1936 the surgeons of the Infirmary tendered Dr. Dixon a testimonial dinner at the Century Club. He spoke touchingly of the past, recalling that he had seen the passing of twenty surgeons, most of them in their prime. To his list there were to be added seven more. He had come to the Infirmary in what he termed its golden age, when Dr. Henry D. Noyes was the executive surgeon. Dr. Noyes it was who furnished the intellect, while Dr. Richard H. Derby, through his friends, supplied the endowments.

Dr. Dixon was a member of the county and state medical societies, the New York Roentgen Society, the American Medical Association, the Associated Roentgenologists of the State of New York and the American Academy of Ophthalmology and Otolaryngology. He was a fellow of the New York Academy of Medicine and of the American College of Radiology.

On his ninetieth birthday, on June 18, 1943, a banquet was held in his honor by the New York Roentgen Society, of which he had been president in 1928-1929. A great cake, set with ninety candles, decorated his table. He had lived so long that it took many blows before he put out the last candle.

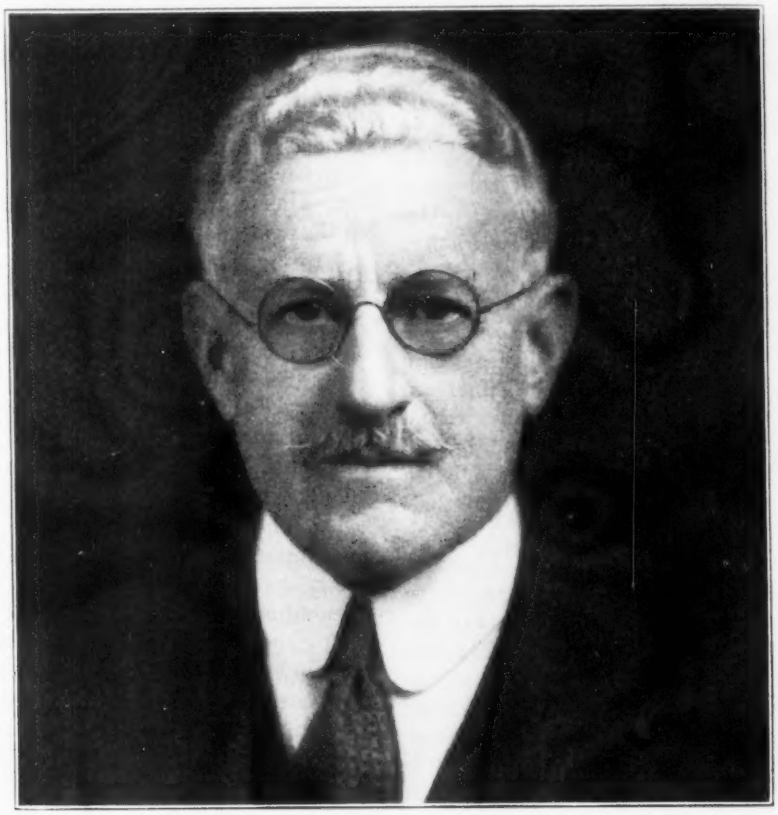
When he was well into his ninety-second year, death claimed him on Oct. 16, 1944, in New York. He must now leave behind the friend of like age, John Elmer Weeks. It has been said that a man's life should be measured, not by years, but by what he has brought forth. From either viewpoint here was a man whose life had been ample indeed.

BERNARD SAMUELS.

JAMES THORINGTON, M.D.

1858-1944

Dr. James Thorington, who died at his home in Philadelphia on Oct. 27, 1944, in his eighty-seventh year, was of Protestant Irish stock, his grandfather having come to Wilmington, N. C., after the War of 1812 and moved successively to Montgomery, Ala. and Davenport, Iowa. During 1837-1839, Dr. Thorington's father, who had studied law at Alabama University, joined a fur company at St. Louis, visiting the Columbia River and meeting Kit Carson on the plains.



JAMES THORINGTON, M.D.
1858-1944

James Thorington was born at Davenport, Iowa, on June 6, 1858, the son of James and Mary Ann (Parker) Thorington. On his mother's side he was descended from Abel Titus and Benjamin Parker, of Vermont, who fought in the Continental Army during the Revolution.

Dr. Thorington's father had served as representative from Iowa in the Thirty-Fourth Congress and later was appointed American consul to Aspinwall, Colombia. In 1874 young Thorington, then a student at Princeton Preparatory School, voyaged around South America by steamer through the Strait of Magellan, from Colon to Panama.

Dr. Thorington graduated from Jefferson Medical College in 1881. His father being still on the Isthmus, he became resident surgeon to the Panama railroad, holding this position until the French concession for the canal became bankrupt in 1889. In 1885 he married Florence M. Jennings, of Harrisburg, Pa. Both had yellow fever and survived. The cause of this disease was not then known, but Dr. Thorington introduced the use of cocaine in its symptomatic treatment (*Am. J. M. Sc.* **99**:150, 1890). Owing to the lawlessness of the time, Dr. Thorington would make his rounds armed with a pistol and carrying a lantern, his identification card

also bearing his name in Chinese characters. In 1884 Dr. Thorington went to Paris to see Ferdinand de Lesseps, who was a friend of the family.

In 1890 Dr. Thorington settled in Philadelphia, where ophthalmology became his immediate interest. By May 1892 he had worked for sixteen months in Dr. Charles A. Oliver's clinic at Wills Eye Hospital and thereafter served as assistant surgeon under Dr. Samuel D. Risley, until May 1900.

Dr. Thorington, after serving as instructor, became adjunct professor of diseases of the eye at the Polyclinic Hospital (1895); when a fourth chair was created, in 1900, he was elected to full professorship. Ursinus College conferred on him the honorary degree of master of arts in 1894. He was oculist to the Vineland (N. J.) Training School, to the Methodist Orphanage and to the Elwyn Training School for Feebleminded, Elwyn, Pa., and for several years lectured on the anatomy and physiology of the eye at the Central Manual Training School.

He was a fellow of the American College of Surgeons and of the College of Physicians of Philadelphia and was a member of the American Ophthalmological Society, the American Medical Association and the Philadelphia County Medical Society. In 1911 he became ophthalmologist to the Presbyterian Hospital (emeritus ophthalmologist, 1923).

Refraction was the field in which Dr. Thorington was particularly known. New vistas were opening, and the lectures given to students of the Polyclinic Hospital formed the basis of his first book "Retinoscopy" (1897). It was one of the first books in which the "shadow test" was explained simply and clearly, and six editions appeared by 1911.

His next book, "Refraction and How to Refract" (1899), an even greater success, passed through five editions by 1910 and was translated into Chinese. "Now I cannot read my own book," said the author. Sixty-five thousand copies of his books were sold during his lifetime.

Two other books, "The Ophthalmoscope and How to Use It" (1906) and "Prisms, Their Use and Equivalents" (1913), were eventually combined with their predecessors into "Methods of Refraction of the Human Eye" (1916), the third edition (1939) being edited by his son. Dr. Thorington contributed the section entitled "Ocular Therapeutics" to "Modern Treatment" by H. A. Hare and H. R. M. Landis (*Philadelphia, Lea & Febiger, 1911, vol. 2, p. 809*). During 1909-1911 he was chairman of the committee which fought the passage of the Optometry Bill.

Thorington was a born teacher, continually improving the apparatus of his time, devising an ophthalmoscope, the axonometer, a schematic eye, a series of lenses to simulate unusual errors of refraction, various test cards, an iris-diaphragm light screen for retinoscopy, an 80 foot (24 meter) range for testing corrections for distance and an electric muscle light for testing phorias at the near point. Dr. Welch recalled that, when testifying in a Baltimore court, Dr. Thorington used the surface of a teaspoon to illustrate astigmatism to the jury.

Through his influence, two of his brothers-in-law took up ophthalmology—Dr. John Ellis Jennings, of St. Louis, who later became an authority on color blindness, and Dr. Charles Jennings, of Pittsburgh. Many physicians were among his patients, including Dr. William H. Welch and Dr. Fielding H. Garrison. Will Rogers and Fred Stone were among the stage notables he attended.

Dr. Thorington believed that manual dexterity was important to a surgeon. He used a straight razor, shaving each side of his face with the corresponding hand. He made cataract incisions with either hand, operating in front and behind the patient's head with equal ease. When his son became his assistant in 1921, it was possible for him to relax, after forty years of practice. He was a better than average golfer and played difficult courses in less than 100 until his seventieth birthday, when he discontinued athletics and surgery.

In 1937 the Philadelphia County Medical Society presented him with a certificate commemorating fifty-six years of practice, but he continued actively until December 1939, when he was in his eighty-first year. He was a man great in human kindness and believed profoundly in future reunion. He is survived by his wife, two sons and four grandchildren.

J. Monroe Thorington.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Cornea and Sclera

KERATOCONJUNCTIVITIS CAUSED BY THE MANZANILLO TREE. R. D. HARLEY, Am. J. Ophth. **27:** 628 (June) 1944.

Harley gives the following summary:

"1. A severe keratoconjunctivitis and dermatitis result from the irritating action of the manzanillo or beach apple tree common to the sea beaches in the Caribbean area.

"2. Blepharospasm, conjunctival edema, and denudation of the conjunctival and corneal epithelium are the most characteristic findings.

"3. Treatment for the eyes consists of saline irrigations, cold compresses, anesthetic instillations, and ocular rest. The dermatitis responds to bland wet dressings and calamine lotion.

"4. Patch tests showed that the sap of the tree possesses the most irritative qualities. Sea water and soap and water for the skin are the most effective, practical, and immediate means at hand of combatting the irritant.

"5. Experiments on a rabbit's eye showed that the toxic action of the sap could be modified by the prompt use of saline irrigations.

"6. Recommended prophylactic measures consist of bathing in sea water with the lids open, followed by a thorough cleansing of the skin with soap and water."

W. S. REESE.

PEDICULOSIS CAPITIS AND CORNEAL LESIONS. A. HIRTENSTEIN, Brit. M. J. **2:** 75 (July 17) 1943.

The author draws attention to the frequency of severe corneal lesions associated with pediculosis capitis. The patients were usually young children who were well fed and without apparent signs of general debility. The presence of pediculosis capitis was discovered by chance on the child's admission to the ward. There was no phthiriasis palpebrarum. Conjunctivitis was always present; corneal ulcers were noted in 14 of the 25 patients; marginal keratitis was observed in 8 patients; hypopyon ulcer, in 1 patient, and severe conjunctivitis without corneal lesions, in 2 patients. Twenty patients were under 10 years of age, and 5 were older. The lesion was always unilateral, and the symptoms were burning and pain followed by photophobia and mucopurulent discharge. Bacteriologic examination showed nothing characteristic. The corneal lesions had no unusual features. They were central or marginal, and sometimes they spread around the corneal margin, forming a complete ring of infiltration. The iris was congested, but there were no corneal deposits. The condition was unusual, as it showed little tendency to respond to routine treatment; in fact, it took a long time to heal. Treatment of the eye consisted in phenolization of the ulcer and the use of atropine, mild protein silver, sulfapyridine tablets and protein shock (milk injections).

ARNOLD KNAPP.

General

A STUDY OF A SELECTED GROUP OF WOMEN EMPLOYED ON EXTREMELY FINE WORK. I. MANN and D. ARCHIBALD, Brit. M. J. **1:** 387 (March 18) 1944.

In this well written and interesting article, the authors report a study of the symptoms of ocular fatigue after performance of fine work for long periods. The group investigated consisted of 28 women and girls employed in a factory at sorting spiral filaments for electric light bulbs. The workers were tested for visual acuity, for muscle balance and for stereoscopic vision. They were also interrogated about their general health and other factors to determine the presence of a possible psychologic element which the authors felt was important in the production of asthenopic symptoms. In a study of the reasons that these workers were able to do continuous close work without fatigue, the authors were impressed by the extremely highly developed stereoscopic sense and muscle balance at the near point. These they regard as more important than refraction. Subjects who apparently did not like their work seemed to be persons with muscular defects rather than high refractive errors. For continued close work it is necessary that the person be orthophoric, without hyperphoria, and have well developed stereopsis and corrected visual acuity of 6/6. It should be stated that if the employees showed fatigue during their training period they were not retained.

Finally, psychologically, the authors believe that the safest group are good average people who are not unduly blessed with imagination or highly intelligent. The factors on which fitness for the work should be based, in order of their importance are: (1) binocular balance, (2) refraction and (3) psychologic stability.

ARNOLD KNAPP.

General Diseases

DERMATOLOGIC LESIONS ABOUT THE EYES. O. S. ORMSBY, Am. J. Ophth. **26:** 850 (Aug.) 1943.

Ormsby describes ocular pemphigus: ectodermosis erosiva pluriorificialis, the triple symptom complex, avitaminosis (ocular), contact dermatitis (dermatitis venenata), xanthelasma, pseudoxanthoma elasticum and associated angioid streaks and lupus erythematosus (of the conjunctiva and lids). W. ZENTMAYER.

EYE MANIFESTATIONS OF XERODERMA PIGMENTOSUM. A. B. REESE and I. E. WILBER, Am. J. Ophth. **26:** 901 (Sept.) 1943.

The authors state that xeroderma pigmentosum, although primarily a disease of the skin and mucous membranes, is frequently marked by ocular complications. They give the following summary of their article:

The cases of 4 patients with xeroderma pigmentosum with ocular complications are reported, including the results of microscopic examination of one eye in which an epithelioma of the cornea developed at the site of an exposure ulcer. One patient died at the age of 7 years and 1 at the age of 11 years, but the other 2 patients have survived up to the time of writing, to the ages of 16 and 30 years. Three of the patients were siblings. From the ophthalmologic standpoint, the disease affects primarily the lower lid, producing an atrophy which begins at the margin and gradually progresses, so that ultimately the entire lower lid may disappear. As a result, exposure of the lower part of the bulbar conjunctiva and cornea produces inflammatory changes, which may lead to symblepharon and corneal ulceration. The lesions that characteristically affect the skin elsewhere involve that of the lids and, to a lesser extent, the conjunctiva.

The article is illustrated. W. ZENTMAYER.

General Pathology

SENILE AND OTHER PIGMENTARY CHANGES IN THE RETINAL GANGLION CELLS. A. LOEWENSTEIN and I. S. MCGREGOR, Brit. J. Ophth. **27:** 486 (Nov.) 1943.

The authors summarize their studies on the changes in the ganglion cells in normal eyes of persons from different age groups and with various diseased conditions.

With the method of examining the retina in bulk, both unstained and stained with scarlet red, they observed that the ganglion cells of the periphery of the retina and the retinal glia cells showed fatty droplets in the plasma in persons with diabetes, hypertension and other diseased conditions, as well as in senile persons. The plasma of the large ganglion cells stained diffusely brownish yellow with scarlet red in patients with hypertensive and diabetic retinopathy and other diseased conditions, and in senile persons. There was no quantitative relation between the fatty droplet content and the diffuse brownish yellow staining.

Unstained retina cleared in glycerin revealed a sulphur yellow pigment in the plasma of the ganglion cells in old age. This pigment is also seen in the retinas of younger persons with diseased eyes, and is analogous to that observed in cerebral tissue in senile and diseased brains. The chemical nature of the diffuse pigment is unknown, but it may be a lipid precursor.

The article is illustrated. W. ZENTMAYER.

Hygiene, Sociology, Education and History

TEACHING AND RESEARCH IN OPHTHALMOLOGY. JOHN HERBERT PARSONS, Brit. M. J. **1:** 430 (March 25) 1944.

To clear confusion of thought as to the role of teaching and research which he believes to exist among ophthalmologists, Parsons first refers to the general principles of medical education. Primarily the functions of the medical man consist in the alleviation and prevention of bodily ailments, and secondarily in research. The author regards ophthalmology essentially as a postgraduate subject and believes that the undergraduate instruction should consist only in what is necessary for the general practitioner to know. Without dealing more fully with postgraduate teaching, Parsons states the belief that medical ophthalmology should receive more attention than it does at present and that the instruction can best be carried out in a hospital for ophthalmic disease associated with a general hospital. He considers ophthalmic research under two headings: (1) clinical research, which has long flourished and should be encouraged in every way but does not need to be under the control of a whole time professor, and (2) basic research, which, on the other hand, requires much knowledge in many branches of pure science and familiarity with laboratory technic, attainments rarely to be found among ophthalmologists. The author does not think that the lavish endowment of whole time professorships solves this problem. In his opinion, it is much more desirable, first, to find the man and, then, to give him the opportunity of close association with other research workers and adequate facilities. For the one who undertakes basic research in ophthalmology, it should be made an important condition that he obtain clinical experience in an ophthalmic center.

The views of one who is an authority both in clinical ophthalmology and in research will well repay careful study and thought.

ARNOLD KNAPP.

Injuries

TREATMENT OF PENETRATING WOUNDS OF THE ANTERIOR SEGMENT OF THE EYE. R. A. CHAVIRA, An. Soc. mex. de oftal. y oto-rino-laring. **17:** 181 (Nov.-Dec.) 1942.

The author describes in detail his treatment of patients presenting such lesions, both at the Juárez Hospital and in his private practice.

In the treatment of wounds of the lid, brow or neighboring regions of the eyes, in order to avoid disfiguring scars, intradermal sutures, reenforced by coaptation stitches, should be used.

With contusions of the eyeball the ocular tension is seldom raised. Mydriasis is usually present, although miosis may occur. Traumatic cataract and retinal detachment may be present as complications, the appropriate treatment for which is given.

Rupture of the sclera usually occurs near the canal of Schlemm (the weakest place) and extends upward and inward; it is due to impact of the globe against the bony orbital brim, and not to the direct blow itself, which usually is received from without and below. The author discusses theories of other investigators as to the mode of production of such tears. When the lesion is so serious that the globe must be condemned, he usually performs exenteration. If the eye is to be saved, he uses the following procedure: surgical cleansing of the wound and adjoining region; ablation or reduction of the prolapsed iris, according to conditions; suture of the scleral wound and use of a conjunctival flap as covering; instillations of atropine and application of a merthiolate ointment, 1:5,000; administration, in some cases, of tetanus antitoxin; binocular bandage and rest in bed; intramuscular injections of milk on succeeding days, and, finally, intravenous injections of mercuric cyanide.

With direct lesions of the sclera and cornea, there is always the possibility of the injury's being complicated by a foreign body. Besides the general complications, traumatic psychoses and neuroses may follow such injuries.

In cases of penetrating wounds of the anterior segment one should (1) operate when the condition is urgent; (2) carefully disinfect the wound and the ocular and periocular regions; (3) secure exact apposition of the margins of the wound, either by suturing or by covering with a conjunctival flap or both, and (4) strive always for conservative treatment.

With serious lesions, when enucleation is indicated, the author advises against immediate operation; rather he recommends a wait of two or three days, until both the surgeon and the patient are convinced that it is the only procedure to follow.

H. F. CARRASQUILLO.

Lens

ABSCESS OF THE CRYSTALLINE LENS. R. O. RYCHENER and E. C. ELLETT, Am. J. Ophth. **26:** 715 (July) 1943.

After the removal of a piece of steel from the center of the lens, a yellowish area, resembling a purulent focus, developed in the anterior substance of the lens. Chemotherapy and paracentesis were followed by alleviation of the infection. Subsequently, secondary glaucoma developed, and this was controlled by corneoscleral trephining and modified anterior sclerectomy. The globe was still preserved three months after the injury, with normal light projection.

Early cultures of the aqueous or of the substance of the lens, obtained by paracentesis with a hypodermic needle, in cases of infection of the anterior segment may aid in determination of the type of medication to be employed.

Chemotherapy in conjunction with fever therapy is suggested as the first step in the medical treatment of all perforating injuries. Repeated paracenteses are of value for abscess of the anterior segment of the globe; they probably induce a higher concentration of the chemotherapeutic agent in the aqueous, in addition to an increased titer of specific antibodies.

W. ZENTMAYER.

SUGAR CONTENT OF CATARACTOUS HUMAN LENSES. P. W. SALIT, Am. J. Ophth. **27:** 612 (June) 1944.

Salit gives the following summary of his studies:

In all, 191 senile cataractous lenses, extracted by the intracapsular method, were analyzed for their sugar content. Significantly, only 0.85 per cent of the incipient cataracts, 2.4 per cent of the mature cataracts and 12.5 per cent of the intumescent cataracts were from diabetic persons. Inclusive of values for diabetic patients, the average sugar content increased from 40.1 mg. per hundred cubic centimeters for incipient cataracts to 47.1 mg. per hundred cubic centimeters for intumescent cataracts and then decreased to 37.1 mg. per hundred cubic centimeters for mature cataracts. Exclusive of values for diabetic patients the sugar content increased from 39.5 mg. per hundred cubic centimeters to 42.2 mg. per hundred cubic centimeters, and then decreased to 35.1 mg. per hundred cubic centimeters respectively. The average value for the 6 cataracts from diabetic patients was 103.1 mg. per hundred cubic centimeters. The intumescent cataract is therefore intimately associated with general disturbances in sugar metabolism.

The average sugar content of the crystalline lens, exclusive of values for diabetic persons, decreased rather uniformly with age from 40 mg. per hundred cubic centimeters, in the fourth and fifth decades, to 36.2 mg. per hundred cubic

centimeters, in the eighth decade. On the other hand, the sugar content of the blood increased from 112 mg. to 145 mg. per hundred cubic centimeters, respectively.

W. S. REESE.

CATARACT EXTRACTION. T. H. BUTLER, Brit. J. Ophth. **27:** 495 (Nov.) 1943.

This article was given as a lecture during the course for the diploma of ophthalmology at the University of Oxford.

The operation of cataract extraction is considered under the heads of the surgeon, the environment, preparation of the patient, the operation preliminary to iridectomy, the extraction, akinesis, difficulties in delivery, loss of vitreous, collapse of the cornea, after-cataract and discission, infection, maturity, bilateral cataract, unilateral cataract, refraction after extraction and alternative methods of extraction. The author's own methods have crystallized down to the old-fashioned operation that was performed at St. Bartholomew's Hospital by Power and Vernon. Butler performs a preliminary iridectomy whenever he can: He uses akinesis, but not retrobulbar injection, because it may produce intraorbital hemorrhage and make the eye soft. A medium-sized conjunctival flap is formed. The speculum is removed before the capsulotomy. If visible cortex remains, as much as will come out easily is expressed. In cases of bilateral cataract, removal of the second cataract is never urged. He thinks that the intracapsular operation is far more dangerous than extraction after capsulotomy. He has done a few intracapsular operations and will never do any more.

W. ZENTMAYER.

Lids

TRANSCONJUNCTIVAL SLIDING-GRAFT TECHNIQUES FOR EYELID RECONSTRUCTION. H. S. SUGAR, Am. J. Ophth. **27:** 109 (Feb.) 1944.

Sugar reviews several methods of blepharoplasty and describes his own technic. This is based on four principles: (1) splitting of the lid into tarsoconjunctival and skin-muscle layers; (2) use of Imre sliding cutaneous flaps, with Burow's triangles; (3) use of the Wheeler method for the formation of lashes, (4) use of the Wheeler "halving" principle. Three cases are reported.

W. S. REESE.

Methods of Examination

ROENTGENOGRAPHY OF EXOPHTHALMOS WITH NOTES ON THE ROENTGEN RAY IN OPHTHALMOLOGY. R. L. PFEIFFER, Am. J. Ophth. **26:** 928 (Sept.) 1943.

In a roentgenographic study of 200 consecutive cases of exophthalmos, it was found that in 138, or about 70 per cent, the evidence was of diagnostic aid. In 84, or 42 per cent, the findings were indicative or diagnostic of the condition. In an additional 20 per cent the changes, when considered in the light of all the clinical and laboratory evidence, were suggestive. The roentgenographic evidence was of greatest usefulness with six of the twelve most common conditions (one third of all the cases)—that is, retention cyst, meningioma, craniostenosis, neurofibromatosis, deformity of the orbit and glioma of the optic nerve. In cases of pseudotumor, hyperthyroidism and oculomotor paralyses, totaling 37, or nearly one fifth of the entire series, the roentgenograph was almost uniformly of no help. In 10 per cent of all cases the exophthalmos was associated with cancer, and in only 13, or 6 per cent, of these cases was the growth a primary intraorbital malignant neoplasm—8 cases of carcinoma and 5 cases of mixed tumor of the lacrimal gland. Cases of combined benign and malignant tumors and cysts constituted 74, or 37 per cent, of the series. Diagnosis was made from positive roentgenographic evidence in 33, or 44 per cent, of these cases, and in 20 additional cases the findings were suggestive.

The elimination of extraorbital conditions as possible causes of exophthalmos is one of the greatest contributions of roentgenography to the study of cases of proptosis.

W. ZENTMAYER.

Neurology

SYNDROMES OF THE PONTILE TEGMENTUM: FOVILLE'S SYNDROME; REPORT OF THREE CASES. W. FREEMAN, H. AMMERMAN and M. STANLEY, Arch. Neurol. & Psychiat. **50:** 462 (Oct.) 1943.

Three cases of lesions occupying the pontile tegmentum and interrupting to a greater or lesser extent the medial longitudinal fasciculus are reported. A lesion in this location produces paralysis of associated lateral movements of the eyes with preservation of convergence. The extent of the lesion determines whether either eye or both eyes will show this disturbance.

A lesion of the pontile tegmentum may produce the following changes:

1. Paralysis of the sixth and the seventh, and sometimes the eighth or the fifth, nerves on the same side.

2. Anesthesia on the opposite side, varying from loss of the sense of position in the limbs to loss of all forms of sensibility in the limbs and face, according to the extent of the lesion. Sensory dissociation over the face on the side of the lesion is irregular, sometimes touch sense and sometimes pain-temperature sense being more affected, the disturbance depending on whether the main sensory nucleus or the descending spinal root is more involved. On the opposite side touch sensibility is usually preserved, but pain-temperature sense is abolished.

3. With laterally placed lesions, mimetic paralysis on the side opposite the lesion and Horner's syndrome on the same side, and sometimes, if the lesion is as extensive as that in case 1, true cerebellar signs as well.

4. With small lesions, damage to the central tegmental fasciculus, giving rise to myoclonus of the palate and sometimes the larynx, the corner of the mouth and even the diaphragm, usually on the side opposite the lesion. This symptom does not seem to occur in cases of extensive damage, as reported in this paper.

5. Reduction of hearing due to damage to the auditory centers or their tracts. If the lesion is in the caudal part of the pons, the loss of hearing is ipsilateral; if the lesion is in the cephalic part, the greater loss is contralateral. Taste sensibility needs further investigation.

R. IRVINE.

Ocular Muscles

SURGERY OF THE INFERIOR OBLIQUE AT OR NEAR THE INSERTION. J. W. WHITE, Am. J. Ophth. **26:** 586 (June) 1943.

In all cases in which operation on both inferior oblique muscles is indicated, recession of the muscles is carried out to equal or to unequal degrees. The amount of recession to be accomplished corresponds to experience in the recession of other muscles. The results were more accurately judged and more permanent when recession of the oblique muscles, rather than tenotomy or myectomy, was performed. When there is associated convergent or divergent strabismus, the external rectus muscles may be prepared for resection or recession, but before the operation is completed the inferior oblique muscles can be easily engaged and recessed. This combined operation through the one conjunctival incision is proving increasingly satisfactory.

The article is accompanied by illustrations.

W. ZENTMAYER.

Refraction and Accommodation

A NEW CROSS-CYLINDER TEST FOR ASTIGMATIC AXIS WITHOUT USE OF TEST TYPE. W. H. CRISP, Am. J. Ophth. **26:** 571 (June) 1943.

The manner of conducting the test is as follows: Before the eye to be tested is placed the spherocylindric combination already found by other methods; while the patient looks at the chart, the examiner remarks that all the radiating heavy black lines are actually of the same intensity but that when the supplemental lens (cross cylinder) is held before the eye the lines will probably appear unequal in intensity. Usually a 0.12 or 0.25 D. cross cylinder is held in the same relation to the patient's axis as in tests for axis with letters; that is, the plus or minus axis of the cross cylinder is held at 45 degrees to one side or the other of the axis of the cylinder in the trial frame. The patient usually notices a difference in the intensity of the radiating lines on the chart, either in both positions of the cross cylinders or in one of them. The patient is then asked to tell which position of the cross cylinder comes nearer (or "nearest") to making the lines on the chart as a whole look uniform. Equal departure from uniformity is the result to be arrived at. If no change in the axis is necessary, the variation in the intensity of the lines will be equal in both directions. The patient's judgment may be checked by rotating the cylinder in one direction or another and repeating the test to find whether he brings the axis back to its previous position.

W. ZENTMAYER.

Retina and Optic Nerve

THROMBOSIS OF CENTRAL RETINAL VEIN. editorial, Brit. M. J. **1:** 227 (Feb. 12) 1944.

Since Holman and Ploman reported favorably in 1938 on the use of heparin in cases of thrombosis of the central vein of the retina, this drug has been tried out in cases of what is generally a progressive condition. The condition, however, is relatively rare. Huggett and Juler (*Tr. Ophth. Soc. U. Kingdom* **62:** 123, 1942) reported on 7 cases, and Rosengren and Stenström (*Acta Ophth.* **20:** 145, 1942), on 30 cases. Neither report bears out the early hopes; good results are exceptional. In an analysis of the end results in 32 cases in which the treatment was not given, Huggett and Juler found that such little improvement as they noted in their 7 cases in which they employed the treatment was paralleled by the results in the cases of the untreated condition, and they stressed that the administration of heparin is not without risk. Rosengren and Stenström observed improvement immediately after treatment in 21 of their cases, but this was not maintained when the patients were reexamined. These results—and early treatment does not seem to influence them materially—bear out the belief that what passes clinically as thrombosis of the central vein of the retina is frequently a blocking of the vein due to intimal proliferation.

ARNOLD KNAPP.

IMPORTANCE OF TEARS IN THE BARRAGE OPERATION FOR DETACHMENT OF THE RETINA. NURI FELIMI AYBERK, Göz. Klin. **1:** 35, 1943.

The author considers it an error to decide at once not to operate when no tear is discovered, and in such cases he employs the barrage technic. He records 3 cases of long standing in which vision was much improved and was retained at the end of one year.

W. ZENTMAYER.

Therapeutics

SOME USEFUL OPHTHALMIC SULPHONAMIDE PREPARATIONS. I. LLOYD JOHNSTONE, Brit. M. J. **1**: 605 (May 15) 1943.

The author is convinced of the therapeutic efficacy of topical administration of the sulfonamide compounds in ophthalmologic practice, but it is necessary to find a vehicle which will keep the substance in contact with the diseased tissues. Among the best of such preparations are an ointment of sulfathiazole, 10 per cent, in a soft wax base, and sulfacetimide, 10 per cent, in a water-miscible base of the appearance and texture of "cold cream." The local application of both these products is of value in treatment of various types of blepharitis and dermatitis. Dermatitis of the face in a child with phlyctenular keratoconjunctivitis cleared up quickly with the sulfathiazole ointment. Burns of the lids may also be treated with the sulfacetimide cream. The author also speaks of the dramatic effect of sulfapyridine in treatment of ophthalmia neonatorum, in which a small portion of a tablet given with or after each feeding clears up the condition in forty-eight hours. The treatment should be continued for another twenty-four hours. Under these conditions local treatment may be reduced to a minimum.

ARNOLD KNAPP.

SULPHANILAMIDE IN EYE DISEASES. N. BAHADUR, Indian J. Ophth. **4**: 84 (Oct.) 1943.

The author reports cases of trachoma, corneal ulcer, gonococcic conjunctivitis and acute purulent conjunctivitis, in the treatment of which sulfanilamide gave excellent results.

W. ZENTMAYER.

Society Transactions

EDITED BY DR. W. L. BENEDICT

NEW YORK ACADEMY OF MEDICINE, SECTION ON OPHTHALMOLOGY

FRANK C. KEIL, M.D., *Chairman*

WILLIS S. KNIGHTON, M.D., *Secretary*

April 17, 1944

Operative Prognosis of Congenital Cataract. DR. ARNOLD KNAPP.

The gravity of the operation for congenital cataract was impressed on me in a recent survey of 400 cases of detachment of the retina. I found that retinal detachment occurred in 37 cases in which operation had been performed for cataract and that 6 of them had been cases of congenital cataract, or nearly 16 per cent, an unduly large proportion. The explanation lies in the fact that the eye with congenital cataract is defective and is consequently more liable to complications. I am under the impression that atypical forms of congenital cataract and cataracts with signs of degeneration are particularly dangerous

Shapland (*Tr. Ophth. Soc. U. Kingdom* **54:** 176, 1934), in a report of 50 cases of detachment associated with aphakia, in 22 of which needling for soft cataract had been performed, drew attention to the frequency of detachment following this procedure. The detachment occurred after an average interval of twenty-four and six-tenths years. Of 40 cases of congenital cataract in Shapland's series, detachment ultimately occurred in 33.

Moore (*Tr. Ophth. Soc. U. Kingdom* **53:** 486, 1933) gave it as his opinion that removal of a lens, whether by extraction or by discission, predisposed to the development of detachment of the retina, and this in no negligible degree. Detachment occurred more frequently after the needling operation for soft cataract, and Moore was so impressed by the poor prognosis for the procedure in young patients that he expressed doubt as to the wisdom of removal of the lens for lamellar cataract unless the opacity was so dense that the eye was otherwise of little value.

I have recently seen a case in point, that of a man with an atypical nuclear cataract on whom I operated in 1901, when he was 13 years of age. The usual discission, followed by a linear extraction, was performed, without complication, on the poorer eye. Ten years later vision in that eye was lost, as a result of detachment of the retina. His next visit was in 1944; he had been able to do his work for thirty-three years with the aid of the eye which had not been operated on, and then a detachment of the retina had suddenly developed in this eye. This case suggests that the detachment occurs in eyes on which operation has been performed for congenital cataract because of defects in the structure of the eye and that an operation may accelerate the development of the detachment.

DISCUSSION

DR. MARK J. SCHOENBERG: What happens in later years to a person with congenital cataract which has not been operated on? Does detachment of the retina develop?

DR. ARNOLD KNAPP: I know of no statistics on that point, but I believe that my last statement answers the question—such eyes are defective, and a detachment is inclined to develop sooner or later, the occurrence of which may be accelerated by operation.

Exophthalmos in Relation to the Thyroid and the Pituitary. DR. ALBERT D. RUEDEMANN, Cleveland (by invitation).

DISCUSSION

DR. ALGERNON B. REESE: When Dr. Ruedemann speaks on exophthalmos associated with disease of the thyroid, one listens; one does not disagree.

I judge that his concept is the one generally held now. The chief ocular manifestations of disease of the thyroid are retraction of the upper lid, due to stimulation of the sympathetic fibers by the thyroxin, and exophthalmos, which is due to a thyrotropic substance in the anterior lobe of the pituitary. This principle presumably causes water retention in the orbit and produces secondary inflammatory changes in the extraocular muscles and in the orbital tissue. These changes, which stem from the pituitary, are kept under control by the thyroxin. Therefore the thyroxin, instead of actually causing exophthalmos, as has been thought in the past, really tends to prevent it. I judge that Dr. Ruedemann does not necessarily believe that this mechanism for the production of exophthalmos holds for the usual protrusion of the eye, but that the imbalance between the function of the pituitary and that of the thyroid is the cause

of so-called malignant exophthalmos. If so, can he give any idea as to the cause of the nonmalignant exophthalmos?

Dr. Ruedemann offers no treatment directed to the pituitary which he has found of any avail for exophthalmos of either the usual or the progressive malignant type. I am sorry he did not mention roentgen treatment of the pituitary, as I understand he has had some experience with it.

He is of the opinion that malignant exophthalmos should be treated either by insertion of tubes into the orbit for drainage of fluid or by orbital decompression. Am I right in assuming, Dr. Ruedemann, that the malignant form tends to regress in six to nine months and that treatment is directed toward prevention of ulcers resulting from exposure during the peak of the reaction?

DR. ALBERT D. RUEDEMANN, Cleveland: It is still difficult, after seeing many cases of this type of exophthalmos over a period of years, to draw definite conclusions concerning the exact pathologic process in the orbit and in the endocrine system. In the first place, ten to seventeen hormones are known to be associated with the pituitary, and it is difficult to evaluate the function of each, either separately or in combination. It is easy enough to say that treatment of the pituitary has helped a number of patients. At the Cleveland Clinic, my associates and I have treated 150 patients with high voltage roentgen irradiation of the pituitary, but we have also given high voltage roentgen radiation to the eyeball, large amounts of thyroid, strong solution of iodine U. S. P., and rest; yet to date everything we have done has seemed fruitless.

In the second place, one must remember that the orbital edema is purely a fluid edema. It is water in the orbit, and when there is anything extra in the orbit, the eyeball has to get out of the way. The interesting thing is that this type of exophthalmos is entirely different, and is associated with no increase in the basal metabolic rate. As a matter of fact, some patients have a rate as low as — 10 per cent, with few other signs of hyperthyroidism. The exophthalmos is the first sign of the disease. Then there is retraction of the levator palpebrae muscle, followed by orbital edema, which I think is of mechanical origin, due to obstruction of the venous outflow. The resultant stasis edema of the orbit builds up its own vicious circle, producing edema of the lids. At one time we had 6 patients at the hospital who were receiving treatment with thyroxin. One went into thyroid shock and gave a great deal of trouble. The woman in the photograph whose eyeballs came forward was treated with thyrourosol, and we had to decompress the orbits within a few days. In only 1 of the 6 patients treated did the condition seem to be stabilized. There is not a period of transition; the patients do not improve.

I believe that malignant exophthalmos is a disease of polyglandular origin. The thyroid is only one gland in the chain which is upset; if it is operated on, the entire chain goes to pieces. Dr. Crile operated on the adrenals in some patients with this condition; my associates and I did a cervical sympathectomy on a number of them, with little or no benefit to the exophthalmos or to the retraction.

I am not entirely discouraged, for we have experimental work in progress which I think will give a new lead to the understanding of the production of exophthalmos and new hope of a means of getting the eyeball back into the orbit. If the fluid edema can be removed from the orbit, we may be able to reestablish the normal drainage, but to date there is little or nothing to offer if one waits until the exophthalmos has developed. If the patient is seen early, when he has retraction of the levator muscle, medical or surgical treatment should be instituted, or if he is seen directly after operation on the thyroid, treatment should be started immediately, before severe malignant exophthalmos has developed. Treatment must be early. Expectant treatment is poor treatment.

Psychosomatic Factors in Ocular Disease. DR. FOSTER KENNEDY.

A new word appearing in the medical vocabulary occasionally means a new idea. The term "psychosomatic" has appeared only receently. I believe it means a new concept for many members of the medical profession, namely, that mind and body are one and the same thing. The word is synthetic, but it tries truly to marry "soma" and "psyche."

We of the medical profession have all been educated to a pathology of fiber, of structure. If the lesion could not be seen under a microscope, there was no lesion. The microscope, by aiding our eyes, has blinded our minds. It is a truism that one's emotions affect one's circulation. Every novelist knows this, but it is in no book of pathology.

The pathology of "fiber" must be supplemented by the pathology of "forces," of the balance or unbalance of the nervous energies of the organism. Gravity cannot be seen, but it is the dominant force in one's daily living. I have seen cases of acute thyroid and adrenal intoxication occur after emotional catastrophe in war.

The interrelationship of mind and body may be studied through the form projections, the visions, fantoms and ghosts seen by persons with organic disease—for example, a neoplasm of the temporosphenoidal region of the brain. I believe such areas of the brain, primitive "smell" areas, subserve early in life intellectual functions, which are later taken over by higher structures, and

that the memories of infancy are recorded in the phylogenetically older structure.

Occipital cortical discharges are not complex and coordinated, but are merely visual patterning, lacking ideational quality.

Allergic reactions in the central nervous system may occur when the sympathetic nervous system of the organism is triggered by emotion. They may explain some of the stigmas of the saints. Retrobulbar neuritis is far from uncommon as an allergic state and occurs as such a reaction most often when the organism is under emotional storm.

And what of hysteria?

Most physicians have a furtive feeling that hysteria is in some way malingering; it may be, but not "conscious" malingering. It is a mode of protection. When the hysterical person has hysterical blindness, he cannot by his conscious will make himself see. Something has happened to prevent his visual cerebral centers from receiving the visual image. I suspect that this block is effected much in the same way as the ignition of a motor car in motion can be stopped by a radio beam; this stimulus can be started from without by suggestion but can be effective only when the intramural emotional condition is fit to receive and implement the message.

What is called a neurosis of the eye occurs commonly in the manic-depressive state, in persons who often, under direct questioning, deny their being depressed but assert they cannot see well enough to read, or sometimes to work. But a depression which is called a mental illness is really due to a disturbance of the balance between the sympathetic and the parasympathetic energy forces, with the production of a melancholic mood, out of which are crystallized organismal symptoms and visceral complaints, all curable by the new method of electric narcosis.

Ocular Symptoms of Psychogenic Origin. DR. C. P. OBERNDORF.

The paper by Dr. Oberndorf is published in full, with discussion, in this issue, page 443.

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY

WARREN S. REESE, M.D., *Chairman*

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April 20, 1944

Pseudobuphthalmos: Report of a Case. DR. WILFRED E. FRY and DR. LOUIS B. COHEN (by invitation).

A well nourished boy aged 5 years was first seen in December 1939. At that time his mother stated that his right eye had turned in since the age of 2 years, the symptom appearing after convulsions. Examination at this time recorded nothing of significance except for internal squint and amblyopia of the right eye.

He was seen again in May 1942, at which time visual acuity was 20/20 in the left eye, and no pathologic condition was noted in the fundus. When he was again examined, in September 1942, he stated that there had been a steady diminution in vision. Visual acuity at this time was about 1/60; but if he looked rapidly to one side and back again, vision of 6/60 was obtained.

Examination of the fundus revealed a cloud of opacities in the posterior portion of the vitreous of the left eye, which obscured the optic disk and the macula, and several small whitish areas in the retina. No retinal detachment was noted, nor was the pupillary reflex changed. A diagnosis of uveitis was made.

General studies, including tuberculin tests, gave negative results. Mixed treatment, as well as a course of tuberculin therapy was tried, but neither appeared to affect the condition. The boy was admitted to the Wills Hospital on Jan. 14, 1944, at which time the fundus picture was essentially unaltered. While he was in the hospital, a flat retinal detachment developed. All general studies at this time, including a roentgenographic examination of the skull and the orbit, revealed nothing abnormal.

Examination with the corneal microscope revealed the following picture:

The cornea was generally clear throughout; the anterior chamber was deep, and the aqueous was somewhat turbid and contained cells. The lens was clear throughout. There were granules and cells throughout the vitreous chamber.

While the patient was in the hospital, it was observed that several small hemorrhages appeared in the upper temporal retina. He was discharged from the hospital, with the tentative diagnosis of Coats's disease (retinitis exudativa), proliferating retinitis, detached retina, uveitis and massive exudate.

Owing to his defective vision (the right eye was amblyopic and showed no signs of inflam-

mation), the boy was admitted to the Overbrook School for the Blind. Subsequent examination at this institution revealed the following condition:

"With oblique illumination the upper edge of a mass could be seen, which was probably a dislocated lens resting on a detached retina. In the temporal part of the periphery, a streak of hemorrhage could be seen on this mass, which represented organized tissue, probably on the surface of the detached retina."

The diagnosis was uveitis, retinal detachment and dislocation of the lens of unknown origin.

In December 1943 the intraocular pressure was observed to be elevated. There were ciliary injection, ciliary staphyloma and a small amount of hemorrhage in the anterior chamber. The eye was enucleated on Jan. 22, 1944.

The pathologic report was as follows:

"A section of the eye measured 32 by 25 mm. The diameter of the cornea was 10 mm. and the anterior chamber 4 mm. in depth. The lens was subluxated. A tumor filled most of the interior of the eye. This mass was roughly in two parts: first, a uniform layer lining the inside of the globe and varying from 2 to 3.5 mm. in thickness, and, second, an irregular mass in the center of the globe. The general arrangement of the cells was in pseudorosettes. The cells were irregularly rounded, with dense nuclei and little cytoplasm. Tumor tissue invaded the optic nerve and the anterior chamber."

The diagnosis was retinoblastoma, secondary glaucoma and pseudobuphthalmos.

Before exenteration of the orbit was performed, the boy died, as a result of intracranial invasion.

A Corneoscleral Suture for Intraocular Surgery. DR. CARROLL R. MULLEN.

I have used a corneoscleral suture in routine extractions, lens loopings and emergencies associated with cataract operations in my services at the Wills Hospital and the Philadelphia General Hospital since 1939. Because of an established procedure of introducing this suture into the tissues, prompt closure can be made by the surgical assistant without interference in the normal routine of the operator's technic. This rapid closure also tends to prevent loss of vitreous.

In addition, the use of this technic makes possible a uniform removal of the suture, so that there is little danger of reopening the section. Other advantageous features of most cataract sutures are included: double closure of the corneoscleral section, tendency toward more rapid healing, less prolapse of the iris, lessening of postoperative corneal astigmatism and sufficient security that the patient may be allowed early use of the fellow eye. In some cases the patient can safely be allowed out of bed sooner if necessary. Perhaps the chief feature of this suture is the ease of its adaptation to removal of dislocated and subluxated lenses and prevention of escape of vitreous.

The eye is prepared for operation in the usual method. A 000000 black braided silk suture in an atraumatic, ½ inch (1.27 cm.) needle with a three-eighths circle cutting edge is passed through the cornea in the temporal to nasal direction 1 to 1.5 under the upper limbus between 11:30 and 1:30 o'clock. The suture is pulled through until locked by a previously inserted double knot. The direction of the suture is then reversed and passed directly above through the bulbar conjunctiva and into the episcleral tissues 1 to 2 mm. above the limbus. The longer free end is not cut away after removal of the needle. The looped continuous nasal portion is then dressed out of line of the incision, and the short free temporal end is dressed downward over the cornea. This permits an opening for the section.

It is important to note that at the entrance through the cornea the knot prevents further passage. The long strand of suture, after coming from the sclera temporally, remains so that a surgical assistant, who is out of the way of the operator, can make rapid closure of the wound on extraction of the lens. The routine placement of the knot at the temporal end of the corneal bite informs the surgeon that on removal the suture should always be severed nasally so the knot cannot be pulled through the tissue or break open the healing corneoscleral section.

In routine operation for cataract the assistant closes the nasal loop gradually as the extraction is proceeding, so that immediately on completion of removal of the lens there is a firm closure of the incision. The two free ends are then tied firmly, but not tightly, with the closing knot over the scleral surface, Nugent utility forceps being used. Irrigation and toilet of the anterior chamber are performed safely after the suture is tied through either temporal or nasal section openings.

In extraction of a dislocated or subluxated lens the initial technic of suture placement is followed, but the assistant makes rapid closure of the section with traction on the free end of the suture as soon as the incision has been completed. This immediately tends to prevent loss of vitreous. If the operator should use a lens loop, this is inserted through the temporal opening, and the lens is moved into position directly under the cornea. The assistant continues to hold the section closed. The operator then extracts the lens through the temporal opening, the assistant relaxing his hold on the suture just enough to allow the lens to come through, and then makes rapid closure by traction on the long free end of the suture. Com-

plete closure of the suture is done in the customary way.

The suture is usually removed on the seventh day. Surface anesthesia should be complete. The nasal band is cut with sharp-pointed scissors, preferably through the strand exposed above the sclera. The suture is then removed with a wet applicator or a forceps without difficulty.

DISCUSSION

DR. CHARLES R. HEED: Within the past year I have had occasion to operate on 3 patients with secondary glaucoma. Two of the patients had total dislocation of a hypermature cataract in the anterior chamber; the third had partial rupture of the zonule. To this series is added a fourth patient with a high grade of myopia who had a mature cataract, with a rupture of the zonule from 11 to 1 o'clock.

On all 4 patients a loop extraction was performed, and the incision was closed with the single suture, as demonstrated by Dr. Mullen.

The absence of loss of vitreous and prolapse of the iris in these 4 patients I attribute to control of the suture during the operation and to the firm closure when the suture is tied.

DR. WILLIAM ZENTMAYER: The suture which has been described here as a Stallard suture is almost exactly like the one devised by Liégard, in 1913. The latter was brought to the attention of this section shortly after that by Dr. S. Lewis Ziegler. It differs only in having a knot at the end of the suture, which Dr. Mullen rightly considers an important modification. The idea of having a knot at the end of the suture, so that it does not pull through the corneal tissue, was suggested by Dr. E. H. F. Frisch, of Atlantic City, N. J., and was referred to by Dr. E. C. Ellett, in his review of cataract operation stitches, "as most ingenious." So Dr. Mullen's suture is really a combination of the two. If the cataract operation must be complicated with corneal sutures, the kind described is doubtless a good one.

DR. I. S. TASSMAN: I have employed the suture Dr. Mullen described ever since I started the use of sutures in cataract extractions, but I never knew the name. I agree with Dr. Mullen that the knot is an advantage, and it is not difficult to apply. Another advantage is that the entire suture is completed before the section is made, which is a distinct advantage over the sutures that are completed afterward. Lastly, I feel as Dr. Mullen does, that this suture requires the least amount of manipulation in its application.

DR. C. E. G. SHANNON: I have lately employed the suture routinely in my cataract extractions and have found it extremely helpful, especially as it eliminates the threat of loss of vitreous.

Orbital Cellulitis Successfully Treated with Penicillin. DR. H. O. SLOANE (by invitation).

This paper has been published in *The Journal of the American Medical Association*. (**126:** 164 [Sept. 16] 1944).

Traumatic Perforations of the Eyeball: Analysis of Fifty Cases. DR. GLEN G. GIBSON.

The records of 50 consecutive cases of traumatic perforation of the eyeball were reviewed. In 12 per cent of the cases the accident occurred while the patient was chopping wood. A conservative regimen was advocated for all but the most severe injuries. Suture of the wound was considered undesirable if it could be avoided. Careful roentgenographic study was advised, as it is possible to overlook small foreign bodies. Prompt resection of prolapsed uveal tissue was recommended. None of the eyes was removed as a prophylactic procedure against sympathetic ophthalmia. In addition to routine procedures, injection of foreign protein, starting on the day of injury, was advised as the most effective form of treatment. Ninety-three per cent of conservatively treated eyes were saved, and 14 per cent had visual acuity of 6/10 or better.

DISCUSSION

DR. EDMUND B. SPAETH: This presentation on perforating wounds of the globe is one of the best I have ever heard. Its conclusions are cleancut and, even if not wholly concurred with, must be seriously considered. Several points could not be stressed by the author, because of the short time at his disposal. I shall therefore not consider additional factors, but only emphasize further certain points presented by Dr. Gibson.

The high incidence of accidents to the globe incurred by persons while working with wood is unnecessary and deplorable. The percentage which Dr. Gibson cites is not too high. These injuries, because of the impact from a relatively large foreign body, also carry the factor of contusion. It is interesting that recently 3 cases of such injuries were encountered: In 1 case there resulted dislocation of the lens into the anterior chamber without perforation of the globe; in the second case the globe was perforated, and there was almost complete iridodialysis with posterior dislocation of the lens. The prognosis for this eye is still grave. In the third case there was a retrobulbar group of wood particles, and, in spite of a discharging fistula in the superior culde-sac roentgenographic shadow suggesting a retrobulbar foreign body, the diagnosis was not made for several months.

Recently, in consultation, a fourth case was seen, with a sliver of wood driven into, but not through, the cornea.

Hospitalization is necessary. Every traumatized globe with perforation is potentially, and for the first forty-eight hours at least, a lost eye. Hospitalization permits controlled rest, the possibility of detailed, careful, continued study and necessary roentgenographic examinations. Without hospitalization the necessary post-traumatic and postoperative foreign protein therapy cannot be carried out.

Dr. Gibson mentioned two situations in which surgical intervention, and I believe early intervention, is necessary: first, when further herniation of the ocular contents is imminent, and, second, when apposition of the edges of the wound cannot be achieved by a pressure bandage.

I should like to add a third condition—any scleral laceration should be sutured. If the scleral rupture is due to impact and lies over the ciliary body, sympathetic ophthalmia may certainly occur as the result of continued uveal irritation. This is the usual sequence, rather than uveal insult from a single trauma to the uveal tract. If the scleral laceration is the point of entrance and exit for removal of a foreign body, it must be closed surgically and treated with diathermy. An intact overlying conjunctiva is not a protection against fistula of the vitreous, continued bleeding, proliferative retinitis and retinal separation. On the other hand, scleral sutures and immediate diathermy are of assistance in their prevention.

Dr. Gibson mentioned the resection of prolapsed uveal tissue; again, I should like to add, "and the release of the edges of the incarceration after the resection." If the wound is recent and clean, and the iris is not macerated and lies subconjunctivally, there is no reason that one should not attempt to replace the tissue, and even use a basal peripheral iridotomy to facilitate this. After the resection of a prolapsed iris, the edges or pillars of the coloboma of the iris should be pushed free from the edges and from the angles in the lines of the irregular lacerations in which they were formerly incarcerated. This applies especially to corneal trauma with prolapse of the iris through a corneal laceration. It is equally applicable to limbic and nonlimbic scleral laceration with prolapse and incarceration of the ciliary body. In suture of these wounds, one must be careful that a knuckle of uvea is not incarcerated in the scleral incision line or in the scleral laceration.

In cases of perforation with an intraocular foreign body, while it is desirable to remove the foreign body immediately, it frequently is not necessary, and occasionally it is unwise. Nonmagnetic foreign bodies are to be removed if possible, with a clear fundus. Hemorrhage into the vitreous will prevent the use of an endoscope or the ophthalmoscope. Foreign bodies incarcerated in the ciliary body, or entangled in the retina must be carefully studied as to the means of removal to prevent irrevocable damage to the ciliary body or to the retina. Possible double perforations with a foreign body lying extraocularly need considerable time for adequate study and diagnosis. This does not excuse procrastination, but it does grant one adequate time for studying and planning the surgical approach.

In every case of perforation of the globe in which there is a possibility of separation of the rètina pinhole glasses should be worn from the time of immediate hospitalization through recovery. This plan seems to have kept low the incidence of retinal separation. Of course, this observation may not be factual, as so many other safeguards against this complication are being used at present. Nevertheless the precaution seems worth while.

Dr. Gibson mentioned the necessary medical care of these patients. His emphasis is well taken. Several days ago, while speaking with a man in the western part of the state who has had a tremendous experience in industrial ophthalmology, I asked him about the value of sulfonamide compounds in prevention of panophthalmitis following perforation. I hoped to obtain a comparison of the incidence of this complication prior to routine sulfonamide therapy with that since its institution. He said that formerly infection occurred in 40 per cent of cases, but since the routine use of sulfonamide compounds this incidence had been reduced to about 20 per cent. Dr. Gibson's figure for infection is one-half that. Other factors in Dr. Gibson's technic which may have been equally important, however, are adequate preoperative preparation, the celerity and excellency of the surgical manipulation and the type of operation carried out. It is perhaps wise to continue use of the sulfonamide compounds as prophylaxis in the presence of perforating injuries.

Intravenous injection of typhoid vaccine is a proved means of combating intraocular inflammation and, with almost equal certainty, intraocular infection. With such therapy the temperature response should be 103 F., or even more. Therefore the dosage must be definitely outlined, since responses of temperature below 102 F. are not adequate. I am certain that this form of nonspecific protein therapy has been the means of saving many eyes which otherwise would have been lost. I am not sure that boiled milk is as effective.

Dr. Gibson states that in his series no eyes were removed as a prophylactic procedure against sympathetic ophthalmia. The nondevelopment of sympathetic ophthalmia in 47 cases, in which the eyes were retained, apparently substantiates his judgment. There is no doubt that an eye with major trauma so severe that the globe is lost

should be immediately enucleated. At the other extreme, prophylactic enucleation is a fallacy and, if carried out as such, will simply mean the unnecessary loss of many eyes.

The development of sympathetic ophthalmia can be combated to a remarkable degree by the prevention of chronic irritation to the uvea through rational reparative surgery and adequate post-traumatic and postoperative foreign protein therapy, under the circumstances of civilian ophthalmic practice.

Further careful, detailed, repeated examinations should enable one to enucleate the eye, if necessary, in sufficient time to prevent the establishment of the disease in the fellow eye. On the other hand, sympathetic ophthalmia is no respecter of persons. It has appeared after rational intraocular operation, with a single needle perforation of the globe, and after a long period of incubation. It behooves one, therefore, to weigh carefully all the circumstances in each case. One should be careful that consistent conservatism also carries with it scrupulous attention and is not simply a *laissez faire* attitude. The first is laudable; the second, deplorable. Dr. Ernst Fuchs stated, "He who has never seen a case of sympathetic ophthalmia develop in a patient under his care is not extraordinarily lucky; he has simply enucleated too frequently." The occurrence of this complication is, however, tragic. It was my misfortune to see bilateral blindness in an 8 year old child follow limbic penetration by a rosebush thorn. The child was first seen four days after the accident, and sympathetic inflammation was already established in the fellow eye.

To recapitulate this most important part of the discussion, rational, but thorough reparative surgical intervention with the prevention of continued uveal irritation, and adequate post-traumatic and postoperative medical treatment are the *sine qua non* in the prevention of sympathetic ophthalmia.

Dr. James S. Shipman: I take exception to a statement regarding the impossibility of implanting a gold or a bone ball in the presence of an infection. As all know, it is difficult to make a successful implantation under such circumstances. However, sulfonamide powder may be dusted into Tenon's capsule first, the bone or gold ball then inserted and a good-appearing artificial eye obtained, in spite of panophthalmitis or infection of less degree. I had a case in which I was going to remove the eye and do only a simple enucleation. The consultant in this case suggested that I use sulfanilamide powder in Tenon's capsule and implant a bone ball in accordance with the usual Frost-Lang technic. This I did, somewhat skeptically; but, to my surprise, the ball stayed in, with a minimum amount of reaction. I take this one exception to Dr. Gibson's paper. One can, in spite of infection, use an implant of bone, gold or plastic material, whichever is preferred, in Tenon's capsule, and I see no reason that it could not be used in the scleral cup and a much better-appearing and more movable artificial eye be obtained.

Dr. Spaeth, in his excellent discussion of Dr. Gibson's paper, did not mention the possibility of closing, with the atraumatic black silk sutures, extensive lacerations in the sclera. In my experience, this procedure has saved many eyes which otherwise might have been lost. I have seen many eyes saved by this method in which the laceration was through the cornea, with extension well back into the sclera for a distance of 6, 7, 8 or 10 mm. over the ciliary body. If the ciliary body itself has not been cut or injured, and this one must determine by means of a loupe, one can still bring the lips of the scleral wound together by means of the intrascleral sutures and save an eye that otherwise would be lost. This has happened a number of times in my experience, and I have saved not only an eye but useful vision. To do this properly, the operator should have his assistant push away the ciliary body and the choroid with a spatula while he inserts the intrascleral sutures to make certain that none of the ciliary body or choroid is pinched off in closure of the scleral wound.

I have enjoyed Dr. Gibson's paper, and I feel that it is worthy of more discussion than has been given here. I disagree with him on the number of blind eyes that he has saved, which to my mind, and I think he must admit, are potential sources of sympathetic ophthalmia. Indeed, Dr. Gibson apparently has not had many cases of sympathetic ophthalmia in his practice. My teaching has been that a badly injured eye is a potential source of sympathetic ophthalmia; certainly, if it is blind, there is no excuse for leaving it in. It has always been my practice to remove such eyes, and, if possible, to make an implant in the scleral cup, as described by Dimitry. This, I think, gives the ideal result so far as appearance and movability of an artificial eye are concerned. Why should one not evacuate the entire contents of the sclera, making certain that all the uveal pigment is removed, put in a bone, gold or plastic ball, obtain a satisfactory artificial eye and remove forever the possibility of sympathetic ophthalmia, thereby relieving oneself and the patient of the ever present worry of trouble later?

Dr. Glen G. Gibson: I thank Dr. Spaeth for preparing such a detailed and enlightening discussion. I am in complete agreement with every statement he has made.

I am also grateful to Dr. Shipman for the presentation of his point of view. I have a great deal of respect for anything he has to say. Unfortunately, in this case I cannot agree, even though I respect his opinion. His comment proves my opening statement that the problem of

the handling of these cases is controversial, and one in which complete unanimity of opinion cannot be expected.

The case of panophthalmitis in which he successfully used an implant with sulfonamide therapy forces me to mention a case in my experience. In at least 1 case of panophthalmitis, and I think in a second, I inserted a ball implant after removal of the eye in the presence of infection without placing the sulfonamide compound in the capsule, and the procedure was successful. Even though this method is not considered the best technic, it can be used.

I disagree with Dr. Shipman's statement about the removal of all blind eyes. This is one of the important points I wish to make, namely, that often an eye which is blind should not be removed, and to the patient is much more desirable than the wearing of a prothesis. The low incidence of sympathetic ophthalmia warrants the saving of many of these eyes, even though they are blind.

Book Reviews

Archivos de la Asociación para Evitar la Ceguera en México. Vol. 2. Director: Dr. L. Sanchez Bulnes. Pp. 248. Mexico, D. F., Mexico: Gómez Farías, 1944.

In 1943 this association celebrated its twenty-fifth anniversary with a scientific session, at which the papers which appear in this volume were presented.

A report was first given by Dr. Fernando Silva on the activities of the hospital of this association during 1943. There were 14,808 consultations, 1,126 refractions and 599 operations. The hospital has a capacity of eighteen beds. The surgical procedures included 20 enucleations; 175 cataract extractions; 15 dacryocystorhinostomies, and 88 operations for pterygium, 20 for glaucoma, 4 for detachment of the retina, 3 for removal of intraocular foreign bodies and 25 for correction of strabismus.

The following scientific papers were presented: "Keratoconus: Its Diagnosis and Treatment," by Martin I. Green, San Francisco; "Sympathetic Ophthalmitis," by Raúl Arturo Chavíra; "The Urgency of the Gonin Operation," by Marc Amsler, and "Pathogenesis of Punctate Keratitis in Onchocerciasis," by A. Torres Estrada. In cases of punctate keratitis Estrada suggests that the presence of microfilarias in the cornea should be considered. A detailed description is given of slit lamp studies of these corneal lesions. Papers on "Management of the Patient with Cataract," by Ray K. Daily; "Amblyopia Exanopsia," by William Thornwall Davis, and "A Comparative Study of the Diagnostic Procedures of Incipient Avitaminosis A," by M. Puig Solanes, follows. The last author expressed the belief that it is not always possible to diagnose avitaminosis A, as the various tests are not exempt from error. In a paper entitled "Blindness from Leprosy Is Preventable," W. H. Hoffmann and Pedro Ramos Báez stated that the modern treatment by intravenous injections of large doses of chaulmoogra oil has succeeded in curing leprous lesions. If this treatment is begun early enough, leprous infection of the eyes does not need to take place. It is important therefore to watch all patients with beginning leprosy and to remember that cases of such infection are likely to appear in families known to be leprous. In a paper entitled "Sclerociliary Iridencleisis," H. Fernández Isassi states that he prefers del Barrio's modification of the iridencleisis operation. This procedure consists of an incision made 10 mm. from the limbus, at 10 o'clock in the right eye and at 2 o'clock in the left eye. A conjunctival flap is then prepared and separated from the sclera; at 8 mm. from the limbus a tangential incision of 3 mm. is made with the keratome through the sclera. A spatula is introduced, and the pectinate ligament is detached throughout nearly half its circumference. A Himly hook is introduced as far as the pupil, and the iris is withdrawn and cut with the scissors at the root of the sclera. A tunnel is thus formed which conducts the aqueous humor to the suprachoroid spaces. In "Surgical Technic for Correction of Spasmodic Entropion and Senile Ectropion," Luís Sánchez Bulnes, after describing the action of the orbicularis muscle in cases of senile ectropion and spastic entropion, explains his operation, in which he corrects the condition by reversing the action of this muscle. In the operation described in the paper entitled "Freeing of Cicatrix After Operation for Glaucoma," by Archimede Bussaca, edema is produced about the adherent scar by injection; then with a fine, curved scissors an incision is made in the conjunctiva at a distance of 1 cm., and a channel is cut next to the sclera up to the point of the adhesion. A similar procedure is carried out on the other side. In 5 cases in which this method was used satisfactory results were obtained. Then follow papers entitled "Cinematographic Demonstration of Problems in Refraction," by Manuel de Rívas Cherif, and "Hemicyclodialysis in Treatment of Chronic Simple Glaucoma," by Enrique Graue Jr. The latter author recommends hemicyclodialysis, in which one-half the circumference of the iris is detached, according to the method of A. T. Estrada. The author finds that this procedure is particularly satisfactory with chronic simple glaucoma but that it should not be attempted in treatment of inflammatory glaucoma. "The Origin of Hypertension in Glaucoma" is discussed by Manuel Uribe y Troncoso, and "Amblyopias of Medicinal Origin," by M. Márquez. The latter author described blindness which occurs after the administration of certain medicaments in the cure of disease not localized to the visual apparatus. He divides these medicinal agents into two groups: substances which cause visual disturbances by local action, and substances which act in a general way. Thus, some drugs alter the transparence of the refracting media, while others produce lesions in the nervous visual apparatus. Other papers include "Melanotic Tumors of the Eye," by L. V. Espino; "A Case of Surgical Reconstruction of the Eye," by Ramón Castroviejo;

"The Vitreous Body in Intraocular Localization of Cysticercus," by José Sáenz Canales; "Hereditary Night Blindness," by Arthur J. Bedell; "The Successful Effort of the Board of Ophthalmology on the Prevention of Blindness," by D. B. Kirby, and "Trachoma as a Factor in Blindness in Mexico," by J. Palomino Rojas. According to the last author, trachoma in Mexico is a degenerative, and not an inflammatory, disease. It is eminently a chronic disease, of minimal gravity and without complications. While it is extremely contagious, it is not an important factor in the production of blindness in Mexico. R. Pacheco Luna ("Trachoma in Guatemala") states that in Guatemala trachoma does not exist; in the cases which occur there the condition does not advance or progress but takes a favorable course and responds to treatment. The author asks: "Why is trachoma contagious in some countries and not in others?" In a paper entitled "Education in Ophthalmology, the Basic Problem for Prevention of Blindness," Moacyr E. Alvaro states that many cases of blindness are due to negligence of the patient or to ignorance of the attending physician. In Latin America the proportion of ophthalmologists is a great deal less than that in the United States. Therefore, the author believes that medical students should receive proper training in ophthalmology and that they should have the advantages of postgraduate courses and internships in hospitals for ophthalmic disease. Boards of ophthalmology should be created. In the training of medical students, interest should be early awakened in ophthalmology; in this way the student will be familiar with the common diseases which produce blindness and will learn about their prevention.

ARNOLD KNAPP.

News and Notes

EDITED BY DR. W. L. BENEDICT

GENERAL NEWS

The National Society for the Prevention of Blindness, Inc.—A prize of $500 is offered by the National Society for the Prevention of Blindness, Inc., 1790 Broadway, New York 19, for the most valuable original paper adding to existing knowledge about the diagnosis of early glaucoma or the medical treatment of noncongestive glaucoma. This award will take the place of two separate prizes of $250 each which were announced some time ago.

Papers may be presented by any practicing ophthalmologist in the Western Hemisphere and may be written in English, French, German, Italian, Spanish or Portuguese. Papers written in the last four languages should be accompanied with a summary in English.

School for Orthoptic Training.—A six months' course for orthoptic technicians will be given by the George Washington University School of Medicine, Washington, D. C., beginning Feb. 5, 1945. Trainees must be sponsored by certified ophthalmologists. The fee for the course is $250. For further details apply to Dr. Ernest Sheppard, School for Orthoptic Training, the George Washington University School of Medicine, 1335 H Street, N.W., Washington, zone 5, D. C.

SOCIETY NEWS

Pennsylvania Academy of Ophthalmology and Otolaryngology.—At the meeting of the Eastern Pennsylvania Association of Eye, Ear, Nose and Throat Physicians held in Wilkes-Barre, Pa., May 10, 1944, it was decided unanimously to change the name of the organization to the Pennsylvania Academy of Ophthalmology and Otolaryngology.

The newly elected officers of the organization will be found in the Directory.

The Scientific Exhibit, American Medical Association, Philadelphia Session.—Application blanks for space in the Scientific Exhibit at the Ninety-fifth Annual Session of the American Medical Association, to be held at Philadelphia, June 18 to 22, 1945, are now available. Application must be made not later than Feb. 12, 1945. The representative to the Scientific Exhibit from the Section on Ophthalmology is Dr. Georgiana D. Theobald, 715 Lake Street, Oak Park, Ill.

Requests for application blanks may be addressed to her or to the Director, the Scientific Exhibit, American Medical Association, 535 N. Dearborn Street, Chicago 10.

CORRECTION

In the article by Dr. Carlos Weskamp entitled "Bowen's Disease of the Cornea," in the April issue (ARCH. OPHTH. **31:** 310, 1944) ("osteitis deformans") was erroneously inserted in line 3 of the fourth paragraph. The term Paget's disease refers here to the cutaneous disease of the nipple.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Dr. P. Bailliart, 66 Boulevard Saint-Michel, Paris, 6e, France.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the Secretariat, 66 Boulevard Saint-Michel, Paris, 6e, France.

International Ophthalmologic Congress

President: Prof. Nordenson, Serafimerlasarettet, Stockholm, Sweden.
Secretary: Dr. Ehlers, Jerbanenegade 41, Copenhagen, Denmark.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 17 Horseferry Rd., London, England.

Pan-American Congress of Ophthalmology

President: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Executive Secretaries: Dr. Conrad Berens, 35 E. 70th St., New York. Dr. M. E. Alvaro, 1511 Rua Consolacão, São Paulo, Brazil.

FOREIGN

All-India Ophthalmological Society

President: Dr. B. K. Narayan Rao, Minto Ophthalmic Hospital, Bangalore.
Secretary: Dr. G. Zachariah, Flitcham, Marshall's Rd., Madras.

British Medical Association, Section on Ophthalmology

President: Dr. W. Clark Souter, 9 Albyn Pl., Aberdeen, Scotland.
Secretary: Dr. Frederick Ridley, 12 Wimpole St., London, W. 1.

Chengtu Ophthalmological Society

President: Dr. Eugene Chan.
Secretary: Dr. K. S. Sun.
Place: Eye, Ear, Nose and Throat Hospital, Chengtu, China.

Chinese Ophthalmology Society

President: Dr. C. H. Chou, 363 Avenue Haig, Shanghai.
Secretary: Dr. F. S. Tsang, 221 Foochow Rd., Shanghai.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

German Ophthalmological Society

President: Prof. W. Lohlein, Berlin.
Secretary: Prof. E. Engelking, Heidelberg.

Hungarian Ophthalmological Society

President: Prof. I. Imre, Budapest.
Assistant Secretary: Dr. Stephen de Grósz, University Eye Hospital, Máriautca 39, Budapest.
All correspondence should be addressed to the Assistant Secretary.

Midland Ophthalmological Society

President: Dr. W. Niccol, 4 College Green, Gloucester, England.
Secretary: Mr. T. Harrison Butler, 61 Newhall St., Birmingham 3, England.
Place: Birmingham and Midland Eye Hospital.

North of England Ophthalmological Society

President: Mr. John Foster, 45 Park Sq., Leeds.
Secretary: Mr. William M. Muirhead, 70 Upper Hanover St., Sheffield.
Place: Manchester, Bradford, Leeds, Newcastle-upon-Tyne, Liverpool and Sheffield, in rotation. Time: October to April.

Ophthalmological Society of Australia

President: Dr. A. James Flynn, 135 Macquarie St., Sydney.
Secretary: Dr. D. Williams, 193 Macquarie St., Sydney.

Ophthalmological Society of Egypt

President: Prof. Dr. Mohammed Mahfouz Bey, Government Hospital, Alexandria.
Secretary: Dr. Mohammed Khalil, 4 Baehler St., Cairo.
All correspondence should be addressed to the secretary, Dr. Mohammed Khalil.

Ophthalmological Society of the United Kingdom

President: Mr. Charles B. Goulden, O.B.E., M.D., M.Ch., 89 Harley St., London.
Secretary: Mr. Frank W. Law, M.A., M.D., F.R.C.S., 30 Devonshire Pl., London W. 1.

Ophthalmology Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Rd., Bombay 4, India.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14, India.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

Oxford Ophthalmological Congress

Master: Mr. P. G. Doyne, 60 Queen Anne St., London, W. 1, England.
Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.

Palestine Ophthalmological Society

President: Dr. Arieh Feigenbaum, Abyssinian St. 15, Jerusalem.
Secretary: Dr. E. Sinai, Tel Aviv.

Polish Ophthalmological Society

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warsaw.
Place: Lindley'a 4, Warsaw.

Royal Society of Medicine, Section of Ophthalmology

President: Col. F. A. Juler, 96 Harley St., London, W. 1, England.
Secretary: Dr. Harold Ridley, 60 Queen Anne St., London, W. 1, England.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and keep it up to date.

São Paulo Society of Ophthalmology

President: Prof. Moacyr E. Alvaro, Consolação 1151, São Paulo, Brazil.
Secretary: Dr. Silvio de Almeida Toledo, Enfermaria Santa Luzia, Santa Casa de Misericordia, Cesario Motta, St. 112, São Paulo, Brazil.

Scottish Ophthalmological Club

President: Dr. S. Spence Meighan, 13 Woodside Pl., Glasgow, C. 3.
Secretary: Dr. Alexander Garrow, 15 Woodside Pl., Glasgow, C. 3.
Place: Edinburgh and Glasgow, in rotation.

Sociedad Argentina de Oftalmologia

Chairman: Dr. Jorge Malbrán, Buenos Aires.
Secretary: Dr. Benito Just Tiscornia, Santa Fe 1171, Buenos Aires.

Sociedad Oftalmologia del Litoral, Rosario (Argentina)

President: Prof. Dr. Carlos Weskamp, Laprida 1159, Rosario.
Secretary: Dr. Juan M. Vila Ortiz, Córdoba 1433, Rosario.
Place: Rosario. Time: Last Saturday of every month, April to November, inclusive. All correspondence should be addressed to the President.

Sociedade de Oftalmologia de Minas Gerais

President: Prof. Hilton Rocha, Rua Rio de Janeiro 2251, Bello Horizonte, Minas Geraes, Brazil.
Secretary: Dr. Ennio Coscarelli, Rua Aimorés 1697, Bello Horizonte, Minas Geraes, Brazil.

Sociedade de Oftalmologia e Otorrinolaringologia de Rio Grande do Sul

President: Dr. Luiz Assumpção Osorio, Edificio Vera Cruz, Apartamento 134, Porto Alegre, Rio Grande do Sul.
Secretary: Dr. Fernando Voges Alves, Caixa Postal 928, Porto Alegre, Rio Grande do Sul.

Sociedade de Ophthalmologia e Oto-Rhino-Laryngologia da Bahia

President: Dr. Theonilo Amorim, Barra Avenida, Bahia, Brazil.
Secretary: Dr. Adroaldo de Alencar, Brazil.
All correspondence should be addressed to the President.

Società Oftalmologica Italiana

President: Prof. Dott. Giuseppe Ovio, Ophthalmological Clinic, University of Rome, Rome.
Secretary: Prof. Dott. Epimaco Leonardi, Via del Gianicolo, 1, Rome.

Société Française d'Ophtalmologie

Secretary: Dr. René Onfray, 6 Avenue de la Motte Picquet, Paris, 7e.

Society of Swedish Ophthalmologists

President: Prof. K. G. Ploman, Stockholm.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö.

Tel Aviv Ophthalmological Society

President: Dr. D. Arieh-Friedman, 96 Allenby St., Tel Aviv, Palestine.
Secretary: Dr. Sadger Max, 9 Bialik St., Tel Aviv, Palestine.

NATIONAL

American Medical Association, Scientific Assembly, Section on Ophthalmology

Chairman: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Secretary: Dr. R. J. Masters, 23 E. Ohio St., Indianapolis.
Place: Philadelphia. Time: June 18-22, 1945.

American Academy of Ophthalmology and Otolaryngology, Section on Ophthalmology

President: Dr. Lawrence T. Post, Metropolitan Bldg., St. Louis.
President-Elect: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
Executive Secretary-Treasurer: Dr. William L. Benedict, 101–1st Ave. Bldg., Rochester, Minn.

American Ophthalmological Society

President: Dr. S. Judd Beach, 704 Congress St., Portland, Maine.
Secretary-Treasurer: Dr. Walter S. Atkinson, 1 Clinton St., Watertown, N. Y.

Association for Research in Ophthalmology, In

Chairman: Dr. Conrad Berens, 35 E. 70th St., New York.
Secretary-Treasurer: Major Brittain F. Payne, School of Aviation Medicine, Randolph Field, Texas.
Assistant Secretary-Treasurer: Dr. Hunter Romaine, 35 E. 70th St., New York.

Canadian Medical Association, Section on Ophthalmology

President: Dr. Alexander E. MacDonald, 170 St. George St., Toronto.
Secretary-Treasurer: Dr. L. J. Sebert, 170 St. George St., Toronto.

Canadian Ophthalmological Society

President: Dr. R. Evatt Mathers, 34½ Morris St., Halifax, N. S.
Secretary-Treasurer: Dr. Kenneth B. Johnston, Suite 1, 1509 Sherbrooke St. W., Montreal.

National Society for the Prevention of Blindness

President: Mr. Mason H. Bigelow, 1790 Broadway, New York.
Secretary: Miss Regina E. Schneider, 1790 Broadway, New York.
Executive Director: Mrs. Eleanor Brown Merrill, 1790 Broadway, New York.

SECTIONAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

President: Dr. N. Zwaifler, 46 Wilbur Ave., Newark.
Secretary: Dr. William F. Keim Jr., 25 Roseville Ave., Newark.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

Central Wisconsin Society of Ophthalmology and Otolaryngology

President: Dr. L. J. Friend, 425 E. Grand Ave., Beloit, Wis.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. Paul A. Chandler, 5 Bay State Rd., Boston.
Secretary-Treasurer: Dr. Merrill J. King, 264 Beacon St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. D. H. O'Rourke, 1612 Tremont Pl., Denver.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. L. L. Bull, 1215–14th Ave., Seattle, Wash.
Secretary-Treasurer: Dr. Barton E. Peden, 301 Stimson Bldg., Seattle 1.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month except June, July and August.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. J. Sheldon Clark, 27 E. Stephenson St., Freeport, Ill.
Secretary-Treasurer: Dr. Harry R. Warner, 321 W. State St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month from October to April, inclusive.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. M. H. Pike, Midland, Mich.
Secretary-Treasurer: Dr. H. H. Heuser, 207 Devidson Bldg., Bay City, Mich.
Place: Saginaw or Bay City, Mich. Time: Second Tuesday of each month, except July, August and September.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. J. C. Decker, 515 Francis Bldg., Sioux City, Iowa.
Secretary-Treasurer: Dr. J. E. Dvorak, 408 Davidson Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. John H. Burleson, 414 Navarro St., San Antonio, Texas.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville, S. C.

SOUTHWESTERN ACADEMY OF EYE, EAR, NOSE AND THROAT

President: Dr. H. L. Brehmer, 221 W. Central Ave., Albuquerque, N. Mex.
Secretary: Dr. A. E. Cruthirds, 1011 Professional Bldg., Phoenix, Ariz.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. W. M. Dodge, 716 First National Bank Bldg., Battle Creek.
Secretary-Treasurer: Dr. Kenneth Lowe, 25 W. Michigan Ave., Battle Creek.
Time: Last Thursday of September, October, November, March, April and May.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Ray Parker, 218 Franklin St., Johnston, Pa.
Secretary-Treasurer: Dr. J. McClure Tyson, Deposit Nat'l Bank Bldg., DuBois.

STATE

ARKANSAS STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. Raymond C. Cook, 701 Main St., Little Rock.
Secretary: Dr. K. W. Cosgrove, Urquhart Bldg., Little Rock.

COLORADO OPHTHALMOLOGICAL SOCIETY

President: Dr. C. A. Ringle, 912–9th Ave., Greeley.
Secretary: Dr. W. A. Ohmart, 1102 Republic Bldg., Denver.
Place: University Club, Denver. Time: 7:30 p. m., third Saturday of each month, October to May, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. F. L. Phillips, 405 Temple St., New Haven.
Secretary-Treasurer: Dr. W. H. Turnley, 1 Atlantic St., Stamford, Conn.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: William O. Martin Jr., Doctors Bldg., Atlanta.
Secretary-Treasurer: Dr. C. K. McLaughlin, 526 Walton St., Macon.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. F. McK. Ruby, Union City.
Secretary: Dr. Edwin W. Dyar Jr., 23 E. Ohio St., Indianapolis.
Place: French Lick. Time: First Wednesday in April.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. J. K. Von Lackum, 117–3d St. S. E., Cedar Rapids.
Secretary-Treasurer: Dr. B. M. Merkel, 604 Locust St., Des Moines.

KANSAS STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. D. Pittman, Pratt.
Secretary: Dr. Louis R. Haas, 902 N. Broadway, Pittsburg.

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. Val. H. Fuchs, 200 Carondelet St., New Orleans.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Robert H. Fraser, 25 W. Michigan Ave., Battle Creek.
Secretary: Dr. R. G. Laird, 114 Fulton St., Grand Rapids.

Minnesota Academy of Ophthalmology and Otolaryngology

President: Dr. George E. McGeary, 920 Medical Arts Bldg., Minneapolis.
Secretary: Dr. William A. Kennedy, 372 St. Peter St., St. Paul.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. William Morrison, 208 N. Broadway, Billings, Mont.
Secretary: Dr. Fritz D. Hurd, 309 Medical Arts Bldg., Great Falls.

Nebraska Academy of Ophthalmology and Otolaryngology

President: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. John Peterson, 1307 N St., Lincoln.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr B. E. Failing, 31 Lincoln Park, Newark.
Secretary: Dr. George Meyer, 410 Haddon Ave., Camden.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Harold J. Joy, 504 State Tower Bldg., Syracuse 2.
Secretary: Dr. Maxwell D. Ryan, 660 Madison Ave., New York 21.

North Carolina Eye, Ear, Nose and Throat Society

President: Dr. Hugh C. Wolfe, 102 N. Elm St., Greensboro.
Secretary: Dr. Vanderbilt F. Couch, 104 W. 4th St., Winston-Salem.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. W. L. Diven, City National Bank Bldg., Bismarck.
Secretary-Treasurer: Dr. A. E. Spear, 20 W. Villard, Dickenson.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. Paul Neely, 1020 S. W. Taylor St., Portland.
Secretary-Treasurer: Dr. Lewis Jordon, 1020 S. W. Taylor St., Portland.
Place: Good Samaritan Hospital, Portland. Time: Third Tuesday of each month.

Pennsylvania Academy of Ophthalmology and Otolaryngology

President: Dr. Lewis T. Buckman, 83 S. Franklin St., Wilkes-Barre.
Secretary Pro Tem: Dr. Paul C. Craig, 232 N. 5th St., Reading.
Time: Last week in April.

Rhode Island Ophthalmological and Otological Society

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Society Library, Providence. Time: 8:30 p. m., second Thursday in October, December, February and April.

South Carolina Society of Ophthalmology and Otolaryngology

President: Dr. J. L. Sanders, 222 N. Main St., Greenville.
Secretary: Dr. J. H. Stokes, 125 W. Cheves St., Florence.

Tennessee Academy of Ophthalmology and Otolaryngology

President: Dr. Wesley Wilkerson, 700 Church St., Nashville.
Secretary-Treasurer: Dr. W. D. Stinson, 124 Physicians and Surgeons Bldg., Memphis.

Texas Ophthalmological and Oto-Laryngological Society

President: Dr. F. H. Rosebrough, 603 Navarro St., San Antonio.
Secretary: Dr. M. K. McCullough, 1717 Pacific Ave., Dallas.

Utah Ophthalmological Society

President: Dr. R. B. Maw, 699 E. South Temple, Salt Lake City.
Secretary-Treasurer: Dr. Charles Ruggeri Jr., 1120 Boston Bldg., Salt Lake City.
Place: University Club, Salt Lake City. Time: 7:00 p. m., third Monday of each month.

Virginia Society of Oto-Laryngology and Ophthalmology

President: Dr. Mortimer H. Williams, 30½ Franklin Rd. S. W., Roanoke.
Secretary-Treasurer: Dr. Meade Edmunds, 34 Franklin St., Petersburg.

West Virginia State Medical Association, Eye, Ear, Nose and Throat Section

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

Akron Academy of Ophthalmology and Otolaryngology

President: Dr. E. L. Mather, 39 S. Main St., Akron, Ohio.
Secretary-Treasurer: Dr. V. C. Malloy, 2d National Bank Bldg., Akron, Ohio.
Time: First Monday in January, March, May and November.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. B. M. Cline, 153 Peachtree St. N. E., Atlanta, Ga.
Acting Secretary: Dr. A. V. Hallum, 478 Peachtree St. N. E., Atlanta, Ga.
Place: Grady Hospital. Time: 6:00 p. m., fourth Monday of each month, from October to May.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Ernst Bodenheimer, 1212 Eutaw Pl., Baltimore.
Secretary: Dr. Thomas R. O'Rourk, 104 W. Madison St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to March.

Birmingham Eye, Ear, Nose and Throat Club

President: Each member, in alphabetical order.
Secretary: Dr. Luther E. Wilson, 919 Woodward Bldg., Birmingham, Ala.
Place: Tutwiler Hotel. Time: 6:30 p. m., second Tuesday of each month, September to May, inclusive.

Brooklyn Ophthalmological Society

President: Dr. Michael J. Buonaguro, 589 Lorimer St., Brooklyn.
Secretary-Treasurer: Dr. Benjamin C. Rosenthal, 140 New York Ave., Brooklyn 16.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. Walter F. King, 519 Delaware Ave., Buffalo.
Secretary-Treasurer: Dr. Sheldon B. Freeman, 196 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. Douglas Chamberlain, Chattanooga Bank Bldg., Chattanooga, Tenn.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. Vernon M. Leech, 55 E. Washington St., Chicago.
Secretary: Dr. W. A. Mann, 30 N. Michigan Ave., Chicago.
Place: Chicago Towers Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati General Hospital Ophthalmology Staff

Chairman: Dr. D. T. Vail, 441 Vine St., Cincinnati.
Secretary: Dr. A. A. Levin, 441 Vine St., Cincinnati.
Place: Cincinnati General Hospital. Time: 7:45 p. m., third Friday of each month except June, July and August.

Cleveland Ophthalmological Club

Chairman: Dr. Shandor Monson, 1621 Euclid Ave., Cleveland.
Secretary: Dr. Carl Ellenberger, 14805 Detroit Ave., Cleveland.
Time: Second Tuesday in October, December, February and April

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. W. S. Reese, 1901 Walnut St., Philadelphia.
Clerk: Dr. George F. J. Kelly, 37 S. 20th St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. H. D. Emswiler, 370 E. Town St., Columbus, Ohio.
Secretary-Treasurer: Dr. D. G. Sanor, 206 E. State St., Columbus, Ohio.
Place: The Neil House. Time: 6 p. m., first Monday of each month.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. C. B. Collins, 704 Medical Professional Bldg., Corpus Christi, Texas.
Secretary: Dr. L. W. O. Janssen, 710 Medical Professional Bldg., Corpus Christi, Texas.
Time: 6:30 p. m., third Tuesday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. Ruby K. Daniel, Medical Arts Bldg., Dallas 1, Texas.
Secretary: Dr. Tom Barr, Medical Arts Bldg., Dallas 1, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. H. C. Schmitz, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Byron M. Merkel, 604 Locust St., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Cecil W. Lepard, 1025 David Whitney Bldg., Detroit 26.
Place: Club rooms of Wayne County Medical Society.
Time: First Wednesday of each month, November to April, inclusive.

Detroit Ophthalmological Society

President: Dr. Raymond S. Goux, 545 David Whitney Bldg., Detroit 26.
Secretary: Dr. Arthur Hale, 1609 Eaton Tower, Detroit 26.
Place: Club rooms of Wayne County Medical Society. Time: 6:30 p. m., third Thursday of each month from November to April, inclusive.

Eastern New York Eye, Ear, Nose and Throat Association

President: Appointed at each meeting.
Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. Rex Howard, 602 W. 10th St., Fort Worth, Texas.
Secretary-Treasurer: Dr. R. H. Gough, Medical Arts Bldg., Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Houston Academy of Medicine, Ophthalmological and Oto-Laryngological Section

President: Dr. Lyle J. Logue, 1304 Walker Ave., Houston, Texas.
Secretary: Dr. John T. Stough, 803 Medical Arts Bldg., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-laryngological Society

President: Dr. Myron Harding, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from November to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. Edgar Johnson, 906 Grand Ave., Kansas City, Mo.
Secretary: Dr. W. E. Keith, 1103 Grand Ave., Kansas City, Mo.
Time: Third Thursday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. Harold Snow, 614 S. Pacific Ave., San Pedro, Calif.
Secretary-Treasurer: Dr. Oliver R. Nees, 508 Times Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Otolaryngology

President: Dr. M. E. Trainor, 523 W. 6th St., Los Angeles.
Secretary-Treasurer: Dr. Orrie E. Ghrist, 210 N. Central Ave., Glendale, Calif.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:00 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye and Ear Society

President: Dr. Joseph S. Heitger, Heyburn Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. J. W. Fish, 321 W. Broadway, Louisville, Ky.
Place: Brown Hotel. Time. 6:30 p. m., second Thursday of each month from September to May, inclusive.

Lower Anthracite Eye, Ear, Nose and Throat Society

Chairman: Each member in alphabetical order.
Secretary: Dr. James J. Monohan, 31 S. Jardin St., Shenandoah, Pa.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. P. S. Constantinople, 1835 I St. N. W., Washington.
Secretary: Dr. Frazier Williams, 1801 I St. N. W., Washington.
Place: 1718 M St. N.W. Time: 8 p.m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member in alphabetical order.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p.m., second Tuesday of each month from September to May.

Milwaukee Oto-Ophthalmic Society

President: Dr. Leon H. Guerin, 324 E. Wisconsin Ave., Milwaukee 2.
Secretary-Treasurer: Dr. Frank G. Treskow, 411 E. Mason St., Milwaukee 2.
Place: University Club. Time: 6:30 p. m., fourth Tuesday of each month from October to May.

Montgomery County Medical Society

Chairman: Dr. H. V. Dutrow, 1040 Fidelity Medical Bldg., Dayton, Ohio.
Secretary-Treasurer: Dr. Maitland D. Place, 981 Reibold Bldg., Dayton, Ohio.
Place: Van Cleve Hotel. Time: 6:30 p. m., first Tuesday of each month from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. J. Rosenbaum, 1396 Ste. Catherine St. W., Montreal, Canada.
Secretary: Dr. L. Tessier, 1230 St. Joseph Blvd. E., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. M. M. Cullom, 700 Church St., Nashville, Tenn.
Secretary: Dr. R. E. Sullivan, 432 Doctors Bldg., Nashvile, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May

New Haven Ophthalmological Society

President: Dr. William H. Ryder, 185 Church St., New Haven, Conn.
Secretary: Dr. Frederick A. Wiess, 255 Bradley St., New Haven, Conn.

New Orleans Ophthalmological and Oto-Laryngological Society

President: Dr. W. B. Clark, 1012 American Bank Bldg., New Orleans.
Secretary: Dr. Mercer G. Lynch, 1018 Maison Blanche Bldg., New Orleans.
Place: Louisiana State University Medical Bldg. Time: 8 p. m., second Tuesday of each month from October to May.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Thomas H. Johnson, 30 W. 59th St., New York.
Secretary: Dr. Wendell L. Hughes, 131 Fulton Ave., Hempstead, N. Y.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

New York Society for Clinical Ophthalmology

President: Dr. Milton Berliner, 57 W. 57th St., New York.
Secretary: Dr. Benjamin Esterman, 983 Park Ave., New York.
Place: New York Academy of Medicine, 2 E. 103d St. Time: 8 p. m., first Monday of each month from October to May, inclusive.

Oklahoma City Academy of Ophthalmology and Otolaryngology

President: Dr. James P. Luton, 117 N. Broadway, Oklahoma City.
Secretary: Dr. Harvey O. Randel, 117 N. Broadway, Oklahoma City.
Place: University Hospital. Time: Second Tuesday of each month from September to May.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. D. D. Stonecypher, Nebraska City, Neb.
Secretary-Treasurer: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m. dinner; 7 p. m. program; third Wednesday of each month from October to May.

Passaic-Bergen Ophthalmological Club

President: Dr. Thomas Sanfacon, 340 Park Ave., Paterson, N. J.
Secretary-Treasurer: Dr. J. Averbach, 435 Clinton Ave., Clinton, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

Philadelphia County Medical Society, Eye Section

President: Dr. Isaac Tassman, 136 S. 16th St., Philadelphia.
Secretary: Dr. Glen Gregory Gibson, 255 S. 17th St., Philadelphia.
Time: First Thursday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. George H. Shuman, 351-5th Ave., Pittsburgh.
Secretary: Dr. Robert J. Billings, 509 Liberty Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Reading Eye, Ear, Nose and Throat Society

President: Dr. Michael J. Penta, 312 N. 5th St., Reading, Pa.
Secretary: Dr. Paul C. Craig, 232 N. 5th St., Reading, Pa.
Place: Wyomissing Club. Time: 6:30 p. m., third Wednesday of each month from September to July.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. Peter N. Pastore, Medical College of Virginia, Richmond, Va.
Secretary: Dr. Clifford A. Folkes, Professional Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

President: Dr. Frank Barber, 75 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Charles T. Sullivan, 277 Alexander St., Rochester, N. Y.

St. Louis Ophthalmic Society

President: Dr. C. C. Beisbarth, 3720 Washington Blvd., St. Louis.
Secretary: Dr. H. R. Hildreth, 508 N. Grand Blvd., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting, 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmo-Oto-Laryngological Society

President: Dr. Belvin Pritchett, 705 E. Houston St., San Antonio 5, Texas.
Secretary-Treasurer: Lt. Col. John L. Matthews, AAF School of Aviation Medicine, Randolph Field, Texas.
Place: San Antonio, Brooke General Hospital, Randolph Field or San Antonio Aviation Cadet Center. Time: 7 p. m., second Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Roy H. Parkinson, 870 Market St., San Francisco.
Secretary: Dr. A. G. Rawlins, 384 Post St., San Francisco.
Place: Society's Bldg., 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. David C. Swearingen, Slattery Bldg., Shreveport, La.
Secretary-Treasurer: Dr. Kenneth Jones, Medical Arts Bldg., Shreveport, La.
Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. Clarence A. Veasey Sr., 421 W. Riverside Ave., Spokane, Wash.
Secretary: Dr. Clarence A. Veasey Jr., 421 W. Riverside Ave., Spokane, Wash.
Place: Spokane Medical Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. A. H. Rubenstein, 713 E. Genesee St., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. H. Blaisdell, 713 E. Genesee St., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

Toledo Eye, Ear, Nose and Throat Society

Chairman: Dr. E. W. Campbell, 316 Michigan St., Toledo, Ohio.
Secretary: Dr. L. C. Ravin, 316 Michigan St., Toledo, Ohio.
Place: Toledo Club. Time: Each month except June, July and August.

Toronto Academy of Medicine, Section of Ophthalmology

Chairman: Dr. W. R. F. Luke, 316 Medical Arts Bldg., Toronto, Canada.
Secretary: Dr. W. T. Gratton, 216 Medical Arts Bldg., Toronto, Canada.
Place: Academy of Medicine, 13 Queens Park. Time: First Monday of each month, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. S. Bockoven, 1752 Massachusetts Ave., Washington, D. C.
Secretary-Treasurer: Dr. John Lloyd, 1218–16th St. N. W., Washington, D. C.
Place: Medical Society of District of Columbia Bldg., 1718 M St. N. W., Washington, D. C. Time: 7:30 p. m., first Monday in November, January, March and May.

Wilkes-Barre Ophthalmological Society

Chairman: Each member in turn.
Secretary: Dr. Samuel T. Buckman, 70 S. Franklin St., Wilkes-Barre, Pa.
Place: Office of chairman. Time: Last Tuesday of each month from October to May.

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